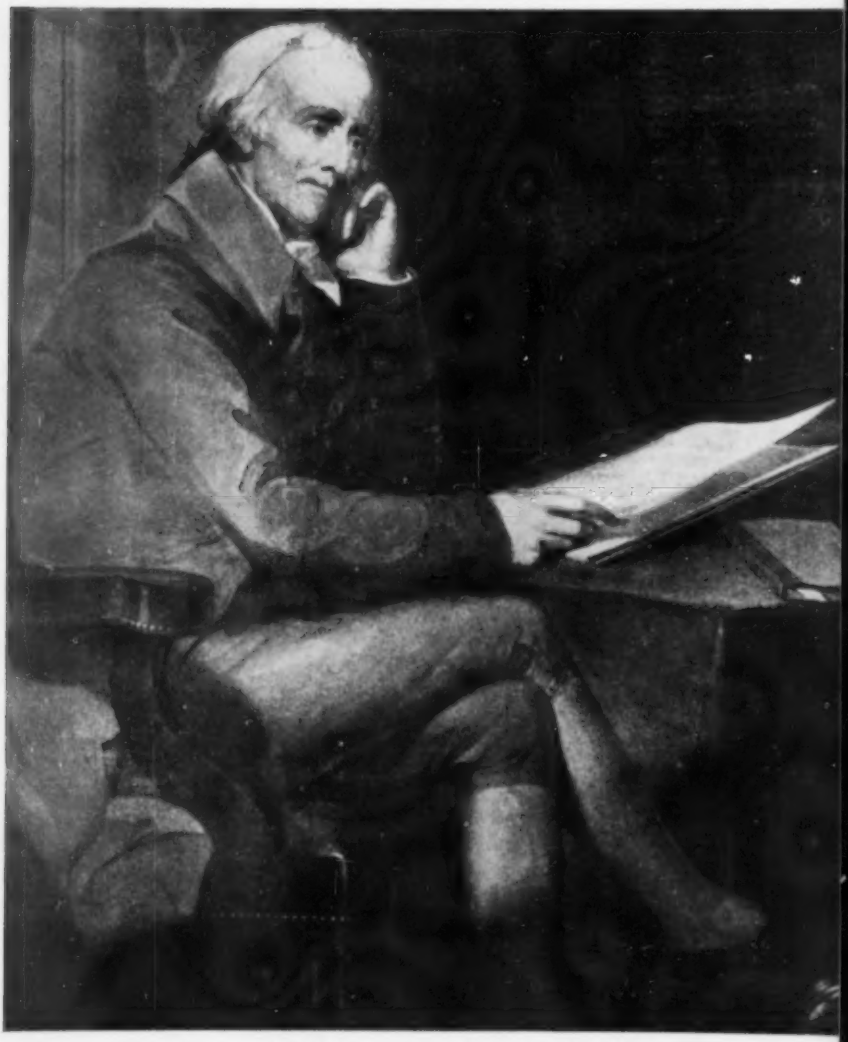
Volume 75

Number 11

ARCHIVES OF PEDIATRICS

NOVEMBER 1958



Pioneers of Pediatrics . . . Benjamin Rush (1743-1813)

Published every month since 1884

WHEN THE PATIENT BECOMES
AN EMPTY CALORIE PROBLEM

bananas

BELONG IN THE DAILY DIET

Bananas are a reliable nutritional ally for younger children and teen-agers, who are notoriously remiss in selecting a well-balanced diet. When these youngsters are tempted to cram themselves with "empty calorie" snacks, the banana fills the gap, helping correct borderline vitamin-mineral deficiencies.

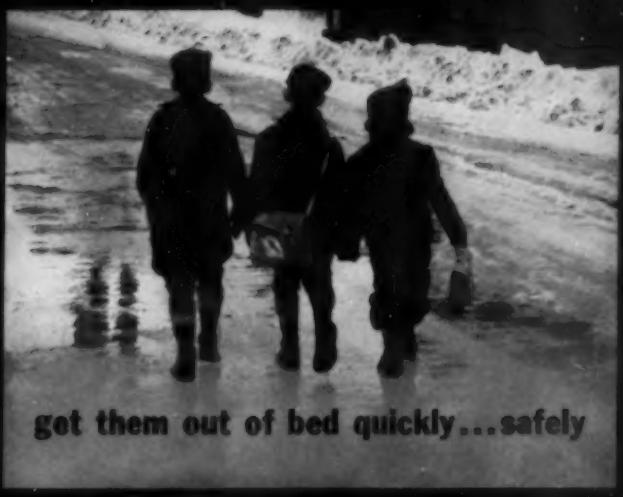
Bananas provide a well-rounded supply of vitamins, and their energy-giving carbohydrates are an aid in keeping pace with the teen-ager's "runaway" metabolism. They can be enjoyed any time, any place — at the table, watching TV, in the back yard, on the playground, or at a picnic.

rich in taste appeal — bananas fill without fattening

- for greater vitality
- for easier weight control
- for better digestion

help yourself to a banana

UNITED FRUIT COMPANY
Pier 3, North River, New York 6, N.Y.



get them out of bed quickly...safely

with tri-sulfanyl

sulfonamide therapy at its best

rapid, maximum recovery assured...
because of rapid, prolonged high blood and tissue levels of triple sulfa mixtures.

worry-free therapy... high urine solubility makes risk of crystalluria virtually negligible. As specific as antibiotics in many infections, but avoids certain of their complications. Danger of moniliasis, gastric upsets, bacterial resistance, sensitivity, blood dyscrasia, etc. reduced to a minimum.

the candy-like flavor of Tri-Sulfanyl syrup appeals to all.

Each 5 cc. of Tri-Sulfanyl syrup, or each tablet contains 0.5 Gm. of total sulfas (equal parts of sulfadiazine, sulfamerazine and sulfathiazole) with 0.375 Gm. of sodium citrate (in syrup only).

4 oz., 16 oz. and gallon syrup; 100 and 500 tablets.

SAMPLES and new literature on request.

arlington-funk laboratories
division of U. S. Vitamin Corporation, 250 East 43rd St., New York 17, N.Y.

ARCHIVES OF PEDIATRICS

Vol. 75 No. 11

November 1958

ALBRO C. GAYLOR, *Publisher*

JOHN FITCH LANDON, *M.D., Editor*

Editorial Board

PHILIP M. STIMSON, M.D., New York
FREDERICK H. WILKE, M.D., New York
P. W. BRAESTRUP, M.D., Denmark
RONALD N. MACLEAN, M.D., Argentina
JOHN ZAHORSKY, M.D., St. Louis
J. FREDERICK EAGLE, JR., M.D., New York
Y. NISHIZAWA, M.D., Japan
ROALD RINVIK, M.D., Norway

Associate Editors

MICHAEL A. BRESCIA, M.D., New York
CATHERINE E. SPEARS, M.D., New Jersey
HERBERT V. VON GAL, M.D., Connecticut

CONTENTS

ORIGINAL ARTICLES

Tuberculosis in Childhood — Part II
Primary Pulmonary Focus and Primary Complex
Philip Schwartz, M.D. 449

Deafness Prevention in Children — Part II
Loss of Hearing for High Tones
Edmund Prince Fowler, Sr., M.D., and Eleanor C. Ronnei, M.A. 474

The Venous Cutdown
Edward G. Stanley-Brown, M.D. 480

Porencephaly and Complicated Cerebral Palsy
Samuel Rosner, M.D., F.I.C.S. 486

Current Literature 490

Copyright, 1958, by E. B. Treat & Co., Inc., 45 East 17th St., N. Y. 3.
All Rights Reserved. Entered as second class matter Feb. 5, 1892, at New York, N. Y., P. O. under the Act of March 3, 1879.

Published monthly since 1884.
Subscriptions: one year—$7.50; two years—$12.00; three years—$16.00.

modern once-a-day sulfa therapy in

NEW CHERRY-FLAVORED KYNEX* ACETYL

*N¹-Acetyl Sulfamethoxypyridazine Lederle

PEDIATRIC SUSPENSION...

In a wide range of infections caused by sulfa-sensitive organisms, delicious new KYNEX Suspension is fully effective. Therapeutic blood levels are rapidly reached and sustained on one easy-to-take dose a day.

Young patients with urinary tract infections, bacillary dysenteries and many other common disorders, respond readily, improve steadily and before long are back on their feet.

DOSAGE IN CHILDREN: Recommended first day dosage is 1 teaspoonful (250 mg.) for each 20 lb. body weight administered immediately after a meal. The dosage for each day thereafter is ½ teaspoonful (125 mg.) for each 20 lbs. administered once a day following a meal.

SUPPLIED: Bottles of 4 fl. oz. Each teaspoonful (5 cc.) of cherry-flavored suspension contains 250 mg. of sulfamethoxypyridazine activity.

LEDERLE LABORATORIES, a Division of AMERICAN CYANAMID COMPANY, Pearl River, New York

Reg. U. S. Pat. Off.

ASYMPTOMATIC ALERT AND ACTIVE

the newest type of

ANTIHISTAMINE

ideally suited for children

"THERUHISTIN"®

Brand of Isothipendyl hydrochloride

SYRUP

2 mg. per 5 cc. (tsp.), bottles of 16 fluidounces

HIGH POTENCY

over 90% effective in 1209 cases of various respiratory and topical allergies (studies included infants, children and adults).[1-3]

DELIGHTFULLY FLAVORED

readily accepted by all youngsters.

LOW SEDATION

99% free of drowsiness in 1209 cases, no other side effects or toxic reactions reported.[1-3]

"exceptionally well tolerated even by infants"

Dosage:
Children, ½ to 1 teaspoonful (1 to 2 mg.) two to four times daily.

Also available:
"THERUHISTIN" Tablets, 4 mg., bottles of 100 and 1,000.
"THERUHISTIN"-S.A. Sustained Action Tablets, 12 mg. per tablet, bottles of 100 and 1,000.

1. New and Unused Therapeutics Committee, Am. Coll. Allergists: Ann. Allergy 16:237 (May-June) 1958. 2. Spielman, A. D.: Ibid. p. 242. 3. Idem: New York J. Med. 57:3329 (Oct. 15) 1957.

Ayerst **AYERST LABORATORIES** New York 16, New York • Montreal, Canada

5880

"Much better-

–thank you, doctor"

COSA-TETRACYN*

GLUCOSAMINE-POTENTIATED TETRACYCLINE

CAPSULES
(black and white)
250 mg., 125 mg.

ORAL SUSPENSION
(orange-flavored)
125 mg. per tsp. (5 cc.), 2 oz. bottle

NEW! PEDIATRIC DROPS
(orange-flavored) 5 mg. per drop, calibrated dropper, 10 cc. bottle

Proven in research

1. Highest tetracycline serum levels
2. Most consistently elevated serum levels
3. Safe, physiologic potentiation (with a natural human metabolite)

And now in practice

4. More rapid clinical response
5. Unexcelled toleration

COSA-TETRASTATIN*

glucosamine-potentiated tetracycline with nystatin

antibacterial plus added protection against monilial superinfection

CAPSULES (black and pink) 250 mg. Cosa-Tetracyn (with 250,000 u. nystatin)

ORAL SUSPENSION 125 mg. per tsp. (5 cc.) Cosa-Tetracyn (with 125,000 u. nystatin), 2 oz. bottle

COSA-TETRACYDIN*

glucosamine-potentiated tetracycline-analgesic – antihistamine compound

For relief of symptoms and malaise of the common cold and prevention of secondary complications

CAPSULES (black and orange) Each capsule contains: Cosa-Tetracyn 125 mg.• phenacetin 120 mg.• caffeine 30 mg.• salicylamide 150 mg. buclizine HCl 15 mg.

Pfizer *Science for the world's well-being*

PFIZER LABORATORIES Division, Chas. Pfizer and Co., Inc. Brooklyn 6, New York

REFERENCES: 1. Carlozzi, M.: Ant. Med. & Clin. Therapy 5:146 (Feb.) 1958. 2. Welch, H.; Wright, W. W., and Staffa, A. W.: Ant. Med. & Clin. Therapy 5:52 (Jan.) 1958. 3. Marlow, A. A., and Bartlett, G. R.: Glucosamine and Leukemia. Proc. Soc. Exp. Biol. & Med. 84:41, 1953. 4. Shallowitz, M.: Clin. Rev. 1:25 (April) 1958. 5. Nathan, L. A.: Arch. Pediat. 75:251 (June) 1958. 6. Cornbleet, T.; Chesrow, E., and Barsky, S.: Ant. Med. & Clin. Therapy 5:328 (May) 1958. 7. Stone, M. L.; Sedlis, A., Bamford, J., and Bradley, W.: Ant. Med. & Clin. Therapy 5:322 (May) 1958. 8. Harris, H.: Clin. Rev. 1:15 (July) 1958.

A5475

new

Tao*

PRONOUNCED TAY-O

(brand of triacetyloleandomycin with glucosamine)

Capsules / Oral Suspension

designed for effective control of common gram-positive infections

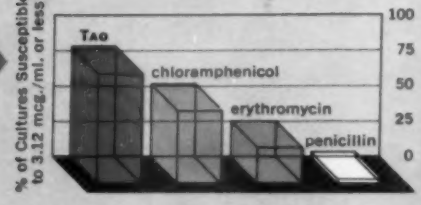
effective . . .

CLINICAL RESULTS	adults	children	all Staph infections
Cured	172 (80%)	148 (89%)	71 (88%)
Improved	28 (13%)	8 (5%)	7 (9%)
Failure	17 (7%)	11 (6%)	3 (3%)

Types of infecting organisms: The majority of identified etiologic microorganisms were Staph. aureus and Staph. albus. Tao has its greatest usefulness against organisms such as: staphylococci (including strains resistant to other antibiotics), streptococci (beta-hemolytic strains, alpha-hemolytic strains and enterococci), pneumococci, gonococci, Hemophilus influenzae.

even against resistant staph[1,2] . . .

Per cent of "antibiotic-resistant" epidemic staphylococci cultures susceptible to Tao, erythromycin, penicillin and chloramphenicol.[1]



well tolerated . . .

REACTIONS:

(a) adults	(b) children
Total—9.2% (20 out of 217)	Total—0.6% (1 out of 167)
Skin rash—1.4% (3 out of 217)	Skin rash—none
Gastrointestinal—7.8% (17 out of 217)	Gastrointestinal—0.6% (1 out of 167)

There was complete freedom from adverse reactions in 94.5% of all patients. Side effects in the other 5.5% were usually mild and seldom required discontinuance of therapy.

plus . . . stability in gastric acid • rapid, high and sustained blood levels • high urinary concentrations • outstanding palatability in a liquid preparation

Dosage and Administration: Dosage varies according to the severity of the infection. For adults, the average dose is 250 mg. q.i.d.; to 500 mg. q.i.d. in more severe infections. For children 8 months to 8 years of age, a daily dose of approximately 30 mg./Kg. body weight in divided doses has been found effective. Since Tao is therapeutically stable in gastric acid, it may be administered at any time, without regard to meals.

Supplied: Tao Capsules—250 mg. and 125 mg.; bottles of 60. Tao for Oral Suspension—1.5 Gm.; 125 mg. per teaspoonful (5 cc.) when reconstituted; unusually palatable cherry flavor; 2 oz. bottle.

References: 1. English, A. R., and Fink, F. C.: Antibiotics & Chemother. (Aug.) 1958. 2. English, A. R., and McBride, T. J.: Antibiotics & Chemother. (Aug.) 1958. 3. Wennersten, J. R.: Antibiotic Med. & Clin. Therapy (Aug.) 1958. 4. Celmer, W. D., et al.: Antibiotics Annual 1957-1958, New York, Medical Encyclopedia, Inc., 1958, p. 476.

Rx NEW YORK 17, N. Y.

Division, Chas. Pfizer & Co., Inc. SCIENCE FOR THE WORLD'S WELL-BEING

*TRADEMARK

Editorial and Business Communications . . .

E. B. Treat & Co., Inc.,
45 East 17th St., New York 3, N. Y.

Subscription:
$7.50 a Year
Foreign: $8.25
Single Copy: $1.00

Original Articles—brief reports of rare and interesting cases or new modes of treatment are solicited. None will be considered for publication except with the distinct understanding that it is contributed solely to this journal. Manuscripts must be typewritten and double-spaced. Please submit only the original copy. The publishers will not be responsible for author's views.

Illustrations—those deemed necessary by the publishers will be furnished free when clear and distinct photographs are supplied. Drawings must be in India ink on white paper.

Copyright—this journal's copyright protects original material; therefore none can be reproduced without publishers' permission.

Reprints—prompt orders for reprints of original articles will be accepted. Schedule of prices will be sent upon request to publishers.

Discontinuances—written notice of discontinuance of subscriptions must be addressed to publishers.

Remittances—payment by check, draft, post office or express money order —please.

Change of address—please give six weeks' notice, with precise name under which subscription is entered, including both old and new addresses and current zone number.

Cover - Benjamin Rush (1743-1813)

Of special significance to pediatrics, Benjamin Rush's paper entitled "An Inquiry into the Cause and Cure of Cholera Infantum" (1773), was noteworthy for its refutation of ideas and opinions that cholera infantum was similar to cholera in the adult—although he did not state the cause of the disease in the adult. Cholera infantum had previously been ascribed to dentition, to worms, and to the eating of fruit during the hot summer. The recording of Rush's treatment of cholera infantum was interesting, but even more interesting was his advice on its prevention . . . such as "the daily use of the cold bath . . . faithful and attentive accommodation of the dresses of children to state and change of air . . . moderate quantity of salted meat occasionally . . . the use of sound old wine in summer . . . cleanliness of skin and clothing . . . the removal of children to the country before approach of warm weather, etc. . . ." Among other noteworthy contributions to pediatrics, were Rush's description of yellow fever, influenza and measles epidemics.

(*"Pioneers of Pediatrics", by Abraham Levinson, M.D.*)

EVERY LISTED PEDIATRIC SPECIALIST

was questioned by an independent research organization about an article published in the Archives of Pediatrics. These specialists were asked whether they agreed with the reprint material.

Of the pediatricians who believed their experience justified an opinion, 156—81.7%—replied yes to all three points in question.

4 out of 5 Leading Pediatricians agree that CREAM OF RICE

NEW, ½ Minute Cooking Time—10 Times Faster!
NEW, Easy-Pouring Spout!

a | **gives "more available caloric energy"**
than any wheat, rye, barley, corn or oat cereal. Of the 227 pediatricians answering definitely, 192—84.6%—said yes.

b | **is "more easily digestible"**
than any other kind of cereal. Of the 248 answering definitely, 212—85.5%—said yes.

c | **gives "nutritional energy more rapidly"**
than any other kind of cereal. Of the 220 answering definitely, 178—80.9%—said yes.

In addition, Cream of Rice is

MOST HYPOALLERGENIC, TOO

As reported in the Archives of Pediatrics by Slobody, Untracht and Hertzmark, "rice . . . shows the fewest allergic reactions of any cereal checked . . . Even children potentially allergic to rice have been shown to tolerate it well when it is cooked in the presence of moisture."

WRITE FOR PROFESSIONAL SAMPLES:
GROCERY STORE PRODUCTS CO., Dept. AP-11
WEST CHESTER, PA.

when sniffles hit the classroom

AND
NASAL CONGESTION
MAKES YOUNGSTERS
MISERABLE

Neo-Synephrine®
HYDROCHLORIDE
Pediatric Nasal Spray
0.25% · 20 cc.
contains Zephiran® chloride 1:5000, antibacterial wetting agent and preservative for greater efficiency

*Designed specifically for children**

Prompt and
Prolonged Decongestion
Sinus Drainage and Aeration

NO STING · NO SEDATION · NO EXCITATION

Plastic Unbreakable Squeeze Bottle
Leakproof, Delivers a Fine Mist

*Also well suited for adults who prefer a mild spray.

Winthrop LABORATORIES • NEW YORK 18, N. Y. • WINDSOR, ONT.
(Formerly Winthrop-Stearns Inc.)

Neo-Synephrine (brand of phenylephrine) and Zephiran (brand of benzalkonium, as chloride, refined), trademarks reg. U.S. Pat. Off.

Kids love to chew

WHITE'S COD LIVER OIL CONCENTRATE TABLETS

Taste as good as candy and provide the average daily requirements of vitamins A and D—PLEASANTLY and ECONOMICALLY.
Each tablet contains vitamins A and D equivalent to one teaspoonful of U.S.P. cod liver oil. Bottles of 100 and 240 tablets.

WHITE'S COD LIVER OIL CONCENTRATE CAPSULES
—whenever *high potency A and D vitamins* are required. Each small capsule provides 12,500 units of Vitamin A and 1,250 units of Vitamin D.
Bottles of 40 and 100 capsules.

Also available: WHITE'S COD LIVER OIL CONCENTRATE DROPS
Bottles of 6, 30, and 50 cc., with dropper.

White's

WHITE LABORATORIES, INC. *Kenilworth, N. J.*

TUBERCULOSIS IN CHILDHOOD

Part II—Primary Pulmonary Focus and Primary Complex in Tuberculosis

By Philip Schwartz, M.D.*

Warren, Pa.

Primary Pulmonary Focus and Primary Complex.

The definition of a pulmonary primary focus and primary complex in tuberculosis was given by Parrot (1876) and his pupils, Hervouet (1877) and Kuss (1898). E. Albrecht, Goldschmied, H. Albrecht, Ghon, Ranke, Redeker, Schürmann, Simon, and Slooff continued these investigations and introduced the concept of a primary tuberculous disease as a characteristic morbid entity into modern pathology. As a result of all these studies—forcefully influenced by experiences in syphilis—the opinion prevails today that the first contact with tubercular bacilli always produces a typical pulmonary focus ("primary focus"), the presence of which, particularly if accompanied by intensely involved regional lymph nodes, proves that a primary infection occurred.

This *primary focus* is generally described as a subpleurally-located, approximately cherry-stone-sized, roundish, concentrically stratified caseous structure, which is subjected to encapsulation and calcification or ossification. It is assumed that foci of this nature always exactly indicate the pulmonary area which was contacted by the inhaled germs at the onset of the infection. Also, it is believed that virulent tubercle bacilli may persist for many years, perhaps even many decades, in a primary focus; that a hematogenic spread may repeatedly originate from an encapsulated primary focus; that an old encapsulated primary focus, by exacerbation of the inflammation, may become the starting point of a chronic pulmonary consumption, and, eventually, that an extinguished, calcified, or even ossified scar of a primary focus may persist for the

* Chief, Department of Pathology, Warren State Hospital, Warren, Pennsylvania; Professor of General Pathology and Pathological Anatomy, University Frankfurt-Main, Germany. Former Director of the Pathological Institute at the University of Istanbul, Turkey.

duration of the whole life. The following assertions also belong to a generally acknowledged, or at any rate, widely accepted teaching concerning the tubercular primary focus: The morphologic aspect of the primary focus is determined only by biological conditions, that is, by reactive properties of an organism invaded for the first time by tubercle bacilli. Thus, a first infection will always produce the same typical primary focus in small children, as in young or aged adults. On the other hand, the presence of a typical primary focus may be accepted as proof of the occurrence of a first infection in an adult of any age—whether young or old. It is maintained that an infiltrate displaying the typical attributes of a primary focus can occur, as a consequence of a first infection, only once in a life, and that all intrusions by tubercle bacilli, occurring later through exogenous infection, produce infiltrates of a different type.

It is accepted that multiple primary foci may develop simultaneously. Also, it is claimed that while a typical small, roundish, concentrically-structured, pulmonary primary focus signifies the effect of a few bacilli, perhaps even of a sole bacillus, a massive first infection may produce voluminous, egg-sized, or sometimes fist-sized, amorphous "atypical" primary infiltrates. Also, cases were repeatedly described in which it seemed that the primary focus was located in the wall of a bronchus ("bronchial primary focus"). Cavities observed in the lungs of small children often were considered to be the result of the softening and dissolution of a large primary infiltrate ("primary focus cavity").

In regard to the *lymph-node involvement* which accompanies the development of a primary focus, the following opinions prevail: The disease of the regional lymph nodes is a phenomenon appearing shortly after the settling of the bacilli within the pulmonary tissue. The volume of the diseased lymph nodes surpasses considerably the size of the primary focus. Correspondingly, we may assume in every case—in children as well as in adults of all ages—in which a small solitary pulmonary focus is associated with large tuberculous lymph nodes, that a primary complex is present. Calcification and, even more, ossification in lymph nodes are characteristic of the primary period; i.e., it is correct to assume that all calcified lymph nodes, detected radiologically in an adult, signify tuberculous foci, which developed in connection with the occurrence of the primary infection. Also, it is generally admitted that tuberculous foci of lymph nodes, developed during the primary period—even if calcified or ossified—for decades contain virulent germs which may be spread under proper conditions by the lymph and blood circulation.

It is thought that the reactivity of lymph nodes, just as the reaction of pulmonary tissue, changes after the occurrence of the primary infection: Post-primary, endogenous, or exogenous infections, causing the development of new pulmonary foci, induce inflammatory processes in lymph nodes which supposedly have a slower and milder course, show no inclination to calcification and—this is particularly emphasized—never produce swellings of a size characteristic of the primary period.

These concepts, although accepted in general, did not remain without dissent. A few years ago, distinguished clinicians pointed out that a pulmonary primary focus often may not be discovered—even in a most careful radiological examination—in instances where large lymph nodes indicate that a fresh exogenous infection occurred. Should these findings not be interpreted in the sense that tubercle bacilli, inhaled or swallowed, may pass through tissues without provoking reactions, only to be eventually retained in parapulmonary lymph nodes where they then exert their pathogenic influence? According to these suggestions, the lymph-node involvement is the primary phenomenon, while the pulmonary focus, or pulmonary foci, develop secondarily, probably in consequence of the discharge of tuberculous lymph nodes into the tracheobronchial system (Kourilsky, Biedermann and Ettedgui). On the other hand, Ghon (1912), Schürmann (1926), Straub (1937), Terplan (1940; 1946; 1051), Blacklock (1947) and Canetti (1950) thought that they observed cases in which no lymph-node involvement accompanied the development of a primary focus. Schürmann (1926), the present writer (1935; 1941) and Terplan (1940; 1946) pointed out that a tuberculous disease can heal biologically so perfectly that a new exogenous infection may produce a typical fresh "primary" (start) complex.

Our own experiences showed that an exogenous inhalatory infection of a person who before this occurrence had not been affected by tuberculosis, always induces the formation of a characteristic pulmonary focus, accompanied by large lymph nodes showing tuberculous infiltration (Figs 1-5).* According to our observations, the pulmonary primary focus displays at the height of its development —approximately 3 to 5 months after the onset of the infection—a pea-sized, roundish center surrounded by a perifocal infiltrate producing a maximum cherry-sized body. Previously, that is, before

* See, also, Figs. 1, 2, 3, 4 of our paper "Lymph-node Tuberculosis A Decisive Factor in Pulmonary Tuberculosis," in Arch. of Pediatrics, 74:159, 201, 1957.

the perifocal infiltrate develops, and later, after the disappearance of the perifocal condensation, the primary focus is so small that it can be found by clinical radiological examination, even in young children, only under particularly favorable conditions, or, perhaps only by chance. All infiltrations larger than a cherry, and definitely those larger than a plum, represent no primary foci, but lymphonodo-bronchogenic aspiration-infiltrations.† We have never encountered "atypical" giant primary foci; neither bronchial primary foci, nor primary cavities. We have not observed multiple primary foci. In cases of typical primary complexes, all other small or voluminous infiltrates accompanying a typically structured primary focus, could be identified in each instance as results of a lymphonodo-bronchogenic superinfection. Remarkably, we repeatedly observed that typical primary foci may be surrounded and covered by aspiration-infiltrations (Fig. 5; also Fig. 4 of our previous paper). No doubt, in these instances the caseation and dissolution of the aspiration-infiltration may cause the traceless disappearance of the primary focus in early stages of the disease.

In numerous other cases we observed the healing of the primary focus: its progressive contraction, encapsulation and incrustation by calcium. *Four years after the occurrence of the infection, the scar of a primary focus may be so small that it can be found only by postmortem radiography* (Fig. 8a). Soon the last trace of the primary focus may disappear and calcified lymph nodes, perhaps containing some recent tubercular infiltrates too, remain as the sole witness of the healing primary tuberculous infection‡ (Figs. 9 and 10). Cherry stone-sized, or larger pulmonary calcium foci appearing in adults as the sole sequelae of healed tuberculosis—or accompanying active processes—represent not scars of a primary focus but remnants of lymphonodo-bronchogenic aspiration-infiltrates. Indeed, hematogenic foci also may calcify.

Tubercular lymph nodes of the primary complex penetrate into the bronchial wall as early as 4 to 6 weeks after the onset of the

† We showed photographs of corresponding cases in our paper published in Arch. of Pediatrics, 74:159 and 201, 1957 (See Figs. 3, 4, and 5).

‡ In a three-year-old girl, who died of diphtheria, we found at autopsy (No. 216/41, Istanbul) a pin head-sized, very hard, calcified primary focus in the right upper lobe, and a pea-sized, very hard calcium focus in a hilum lymph node of the right side. While no other tuberculous pulmonary changes were present, a number of hilum lymph nodes contained tuberculous foci, being mostly in advanced healing. Because of the importance of this observation, we intend to describe it in a separate paper.

Fig. 1. *Primary complex of the left lung. Spreading of lymph-node tuberculosis to the right.* 6-year-old boy, who died of tuberculous meningitis. Endocarditis; subaortic septum defect. (A. 844/47, Istanbul.)

The primary focus, about bean-sized, located in the apex of the left upper lobe, was visible in a radiograph taken one day before death. In this radiograph enlarged tuberculous lymph nodes of the left interlobar space also were represented.

The autopsy disclosed intense and extensive involvement of the parapulmonary lymph nodes on *both* sides. On the right side not only the bifurcation lymph nodes were markedly enlarged and caseated, but also the subclavian paratracheal lymph nodes, as well as other lymph nodes of the intertracheo-broncho-pulmonary space; furthermore, lymph nodes of the superior and inferior interlobar spaces and of the intrapulmonary interbronchial angles. Patient died of meningitis, before lymphonodogenous bronchial lesions could develop.

An egg-sized diffusely caseated lymph-node complex was located between the pancreas head and duodenum. The spleen contained very numerous, up to lentil-sized caseated foci. A fine nodular miliary dissemination was present in both lungs.

We assume that the infection occurred approximately 6-8 weeks before death.

Explanation of the signs see page 472.

infection, discharging into the tracheobronchial system (Figs. 2-5; also Figs. 1-6 in our repeatedly mentioned previous paper). As a consequence of this process, large lymph-node complexes, which were evident in clinical radiographs, may diminish in a surprisingly short time. The complete discharge of necrotic and dissolving lymph nodes explains why sometimes after healing of the primary complex, no calcification remains in the hilar area. However, most often, some calcified foci of lymph nodes persist, which in the course of years gradually shrink and eventually disappear (Figs. 9 and 10b). Later-occuring exacerbation may open the way for tuberculous lymph nodes into bronchi, thus giving them the opportunity—sometimes many years after the healing of the primary complex—to be rid of their calcium foci. Nevertheless, the elimination, or, generally speaking, the absence of calcium deposits, only rarely signifies the definite healing of the lymph-node tuberculosis accompanying primary infection. The more carefully we investigate, the more often will we find somewhere in the organism—in the thoracic or abdominal cavities—tubercular lymph nodes containing bacilli, in children and in adults displaying only extinguished pulmonary remnants of a tuberculous condition. At any rate, as in the case of Fig. 8 observed, from a primary complex which originally showed all typical attributes, eventually only a shrinking, encapsulated, caseous primary focus—or its scar—remains, while regional tuberculous lymph nodes or their calcifications are absent. On the other hand, as the case of Fig. 9 proves, because of the traceless disappearance of the primary pulmonary focus, we may observe—even in young children—only tuberculous involvement of the hilum lymphnodes.

The following observations concerning lymph-node involvements in primary tuberculosis merit to be emphasized: In cases in which the primary focus develops in the left lung, the accompanying lymphnode tuberculosis spreads in a surprisingly short time to the right (Fig. 1-3); it may even occur that lymphonodogenous bronchial lesions—and therefore aspiration-infiltrations—appear earlier in the right lung, causing serious misinterpretation of the clinical aspect. On the contrary, if the primary process starts on the right side, there is a great probability that the intrapulmonary lymph nodes of the left lung will remain spared. (Figs. 4 and 5)

In small children in whom the nature of a first infection can be defined without any doubt, the following types and developmental stages of initial foci—start foci—occur: (1) the primary focus appears—approximately 3 weeks after the onset of the infection—as a small, rather shapeless and irregularly limited, caseous pneu-

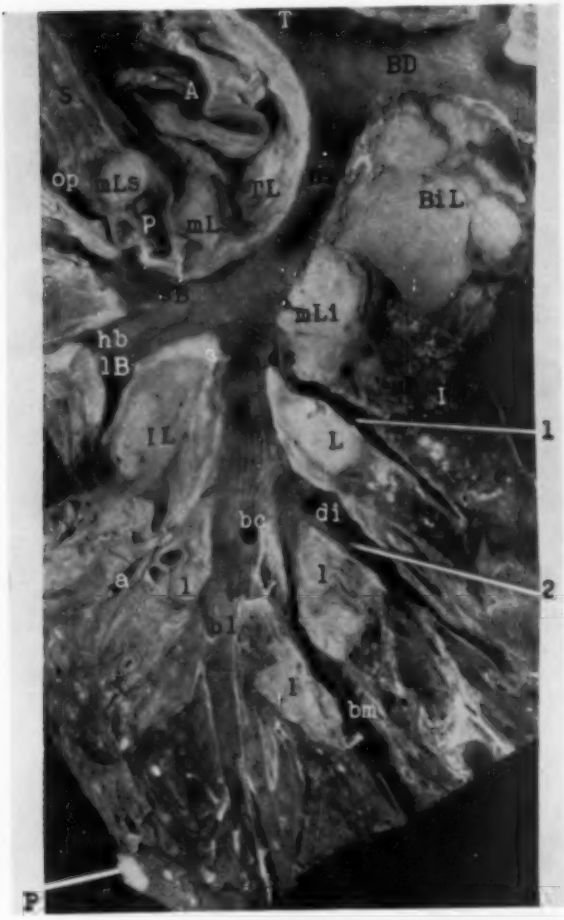


Fig. 2. *Fresh primary complex of the left lung.*

Primary focus (F) located subpleurally in the lateral basal region of the lower lobe. Beginning penetration of intensely caseated interbronchial lymph nodes into the bronchial system. A few small, fresh aspiration-infiltrates of the left lower lobe. Intense swelling and diffuse caseation of very numerous parapulmonary lymph nodes of the right side.

Three-year-old boy, who died of tuberculous meningitis (A. 371/50, Istanbul), before larger perforative bronchial ulcers and more extensive aspiration-infiltrations could develop.

We assume that the infection occurred approximately 7 to 10 weeks before death.

Explanation of the signs see page 472.

monic infiltrate ("amorphous stage of the primary focus"). (2) As early as 4 to 5 weeks after the infection begins, the now approximately cherry stone or pea-sized start focus, surrounded by soft aerated pulmonary tissue, becomes a roundish, although not yet encapsulated, structure ("stage of delimitation of the primary focus"). (3) In 6 to 12 weeks from the start of the infection, a concentric stratification of the primary focus begins to appear, accompanied by a loose calcium incrustation of its necrotic masses; at the same time, the surrounding pulmonary tissue becomes filled with tubercles and with a pneumonic exudate. Thus, the entire primary infiltrate, now approximately cherry-sized, displays two zones: The center contains the cherry stone-sized, roundish, caseous primary focus, which is enveloped in a more-or-less fleshy—at any rate, not diffusely necrotic—perifocal condensation. The tubercles of the perifocal zone developed as a consequence of contact infection, originating from the primary focus and extending along the septal lymph paths; the main mass of the perifocal infiltration is located within the alveoli between the tubercles. This stage ("stage of the perifocal infiltration") represents the highest point in the development of the primary focus. (4) Primary foci observed 5 to 8 months after the infection already had lost its perifocal cover and appear as small, ball-like, caseous bodies, subjected to encapsulation by connective tissue and to a progressive hardening by accumulation of calcium sediments ("stage of encapsulation", "stage of calcification and shrinking"). [See also Sweany]

Endeavoring to recognize the reality, it was necessary to abandon certain deeply rooted concepts concerning the nature of tuberculous inflammation. We mention only a few of these traditional doctrines here: The old conviction that a tuberculous focus can never heal completely; that a pulmonary infiltration caused by tubercle bacilli always will be subjected to necrosis—that is, it will lead always to destruction of the affected tissue; that an inflammatory infiltration caused by tubercle bacilli cannot be resolved without destroying pulmonary tissue; and eventually, that the formation as well as the healing of specific changes—of individual tubercles, conglomerates of tubercles, of caseous pneumonia, of cavitation—are very slow processes necessitating months, years and even decades!

Indeed, without the experience we gained in experimental investigations, it would be perhaps impossible to imagine that a massive caseous pneumonia, almost completely filling a pulmonary lobe and already displaying marked signs of dissolution, may develop as soon as 8 weeks after the onset of an exogenous primary infection;

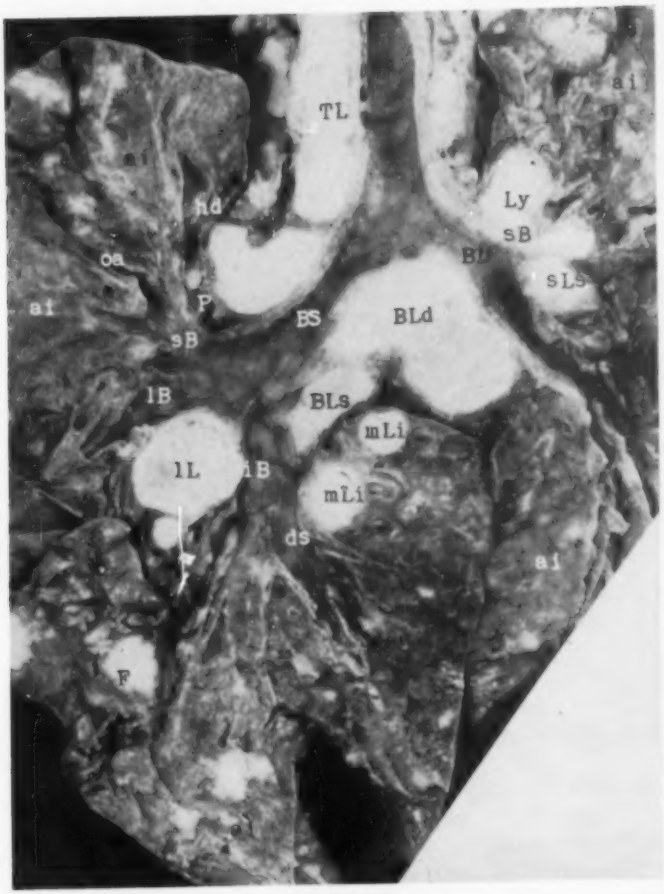


Fig. 3. *Fresh primary complex of the left lung.*

Primary focus (F) located in the superior lateral sector of the left lower lobe, supplied by the axillary bronchus. Intense caseous involvement of lymph nodes in both lungs. Marked stenosis of the trachea and of both main bronchi caused by surrounding large caseous lymph nodes. Very numerous mucosal erosions of the trachea, the main bronchi and of the lobar bronchi bilaterally, following extensive lympho-nodogenous penetration. Very numerous small aspiration-infiltrations in all parts of both lungs. Dense hematogenic dissemination with intense interfocal infiltration.

A 14-month-old boy who died of tuberculous meningitis (A. 239/49, Istanbul), just before the appearance of a very large perforative, ulcerous lesion of the left main bronchus and the carina.

The infection occurred, probably, 8-12 weeks before death.

Explanation of the signs see page 472.

also, that a disintegrative process, as early as 12 to 16 weeks after the occurrence of the primary infection, produces giant, almost completely emptied cavities; that cells which—in a rubber eraser-like infiltrate displaying no necrosis—fill the pulmonary alveoli, contain innumerable tubercle bacilli; and that a primary focus may shrink, within 4 years, into a very minute focus—the presence of which can be shown only by post-mortem radiography—or that a primary focus may even disappear tracelessly this early. It is imperative to keep the facts we just mentioned always in mind when evaluating observations related to pulmonary tuberculosis.

Before we understood the paramount pathogenetical importance of the discharge of tuberculous lymph nodes into the tracheobronchial system, we collected a number of lungs of adults, in which tuberculous foci,—subpleurally-located and sometimes even concentrically stratified—were present, resembling in every respect classic primary lesions in children. In all these cases, the lymph nodes were intensely enlarged and casefied just as in typical primary complexes. These observations were interpreted according to the presence or absence of calcified old pulmonary foci, as "late primary infections", or, as "post-primary start complexes"—that is, as infiltrates resulting from a new exogenous infection after biological healing of a previous disease. This explanation seemed to be the more plausible because, in earlier investigations, we had the opportunity to observe that tuberculosis primary complexes in the stage of advanced healing occur in children only 4 years of age!

We also repeatedly observed pulmonary involvements in adults, resembling primary complexes of small children, during the last few years. However, in these cases, we were able to prove that pulmonary infiltrates suggesting primary foci were produced by the discharge of tuberculous lymph nodes, in which old processes exacerbated. Fortunately, we preserved the anatomical specimens of some of our earlier "late first infections" and "post-primary start complexes," thereby being able to show that those "primary" foci also developed following the discharge of tuberculous lymph nodes into the bronchial system. Once more it was confirmed that we see only what we know and that very obvious facts may be neglected if we do not look for them.

Thus, we confront a completely new situation: *There is no possible doubt that following bronchial lesions induced by exacerbated tuberculous processes in lymph nodes, pulmonary infiltrates may appear in adults and children which perfectly resemble genuine primary foci.* Furthermore, lymph nodes affected by reactivation

of the tubercular inflammation are, at the time they begin to discharge into the bronchial system, often intensely enlarged and diffusely caseated, which also may strengthen the suggestion of the presence of a "late first infection," or of a "postprimary start complex."

All these observations show the necessity of a profound revision of our concepts regarding the nature of pulmonary foci, hitherto

Fig. 4. *Fresh primary complex of the right lung.*

Primary focus (F) showing beginning encapsulation, located subpleurally in the upper dorsal sector of the middle lobe. Ulcerous discharge of an infraarterial, caseated, softened lymph node of the superior lateral interlobar space into the main bronchus of the middle lobe, just before the orifice of the inferior limb of the posterior branch (Probe 2). Extensive, wedge-shaped ("segmental") aspiration-infiltration (ai) of the middle lobe sector, aerated by the inferior limb of the posterior middle lobe bronchus. Beginning disintegration (cav) of the aspiration-infiltration. Primary focus and aspiration-infiltration are quite near to each other. Intense bulging and caseous tubercular infiltration of the carina. Two small perforative defects (probe 1) of the carina, caused by discharge of the intensely enlarged bifurcation lymph nodes. One of these fistulas affects the main bronchus of the left lung and could be functioning as the source of an aspiration-infiltration of the left lung. Otherwise the lymph nodes of the left lung were not involved.

We assume that the discharge of the tuberculous lymph node into the main bronchus of the middle lobe occurred 4-6 weeks before death.

Two-and-a-half-year-old girl, who died of tuberculous meningitis (A. 276/49, Istanbul).

Explanation of the signs see page 472.

considered as tuberculous primary infiltrates. *The presence of these foci does not signify that an organism contacted tubercle bacilli the first time, but that at the time of the contamination, the pulmonary tissue showed no increase of its disposition to tubercular inflammatory reactions, and that the inflammatory properties of the microorganisms were not depressed by substances providing immunity.*

This "normergic" behavior of an organism, as yet not involved by tuberculosis, appears to be self-evident; however, all other infiltrates, which develop during the period of increased sensitivity to inflammatory reactions following new exogenous or endogenous (for example, bronchogenic) infections, are not small condensations resembling typical foci, but much more extensive ones. On the other hand, exogenous infections—or endogenous spread—if occurring during the period of immunity, perhaps induce no pulmonary changes at all. However, in cases in which both intensely increased postinfectious disposition to inflammatory reactions and effective immunity are present simultaneously, we observe the development of very extensive rubber eraser-like, that is, non-necrotizing infiltrations of the pulmonary tissue. Thus, the observation of a characteristic lymphonodo-bronchogenic pulmonary infiltrate resembling a primary focus—in a case in which, following the exacerbation of a chronic lymph-node tuberculosis, a new cycle of the process began—revealed that the lymph node perforation occurred in a period in which immunity had already been exhausted, or suppressed by an intercurrent involvement, and a new increase of the disposition to inflammatory reactions was not yet present. Accordingly, in principle it would be possible that, in a person with hidden virulent bacilli in inactive foci, a new exogenous infection—occurring, perhaps, just as a cycle of the chronic tuberculous process closes—produces an infiltrate resembling a primary focus. However, our new experiences disprove these assumptions. We have observed hitherto only cases of the post-primary period, in which typical start foci appeared following the discharge of tuberculous lymph nodes.

We believe that the occurrences which induce the development of an initial infiltrate resembling a primary focus, in the course of a chronic tuberculous process, could be explained as follows: *The exhaustion of the immunity, induced by natural decrease or by intercurrent influences, permits germs—which had vegetated in lymph nodes as harmless parasites—to proliferate and once more develop their inflammatory properties; perforative lesions of the bronchi appear, followed by small aspiration-infiltrations resembling primary foci.*

Fig. 5. *Fresh primary complex and lymphonodogenic aspiration-infiltrates of the right lung in a 5-month-old boy, who died of "pneumonia."* (A. 25/29, Frankfurt-Main). The autopsy disclosed a lentil-sized, rather hardened, encapsulated primary focus (F) located in the sector of the right lower lobe, aerated by the horizontal branch of the superior dorsal bronchus. A cherry-sized, almost completely caseated aspiration-infiltration (Ai) thoroughly joined the primary focus. This aspiration-infiltrate resulted from the discharge of a cherry-sized infra-arterial lymph node (mLi) of the medio-dorsal interlobar space into the stem of the superior dorsal bronchus. Another more voluminous aspiration-infiltration, (ai), approximately of the same age, and also displaying massive caseation, affected the dorsal segment of the middle lobe; it followed the discharge of an infra-arterial lymph node of the superior lateral interlobar space into the middle lobe main bronchus. Both lungs were filled with a dense hematogenic dissemination and an accompanying fleshy (rubber eraser-like) interfocal infiltration. (See Fig. 7).

We assume that the infection occurred 4-5 months before death and that the aspiration-infiltrations were 2-3 months old.

In spite of the intense tuberculous involvement and considerable enlargement of the parapulmonary lymph nodes of the right lung, those on the left side remained spared! (See Fig. 7).

Explanation of the signs see page 472.

All these pathologic-anatomical observations, and the hypotheses we link with them, make it understandable that in some clinical cases—in children and adults, giving the impression of primary infections—first lymph nodes and only later parenchymal infiltrates become visible. Nevertheless, findings of this kind cannot be considered proof that a primary infection causes the involvement of lymph nodes first, and only secondarily the appearance of parenchymal infiltrates. *A typical pulmonary primary infiltrate is present in every case in which an exogenous infection has been transmitted by the tracheobronchial system.* However, the initial focus is much too small to always be clinically recognized. In contrast, lymphonodo-bronchogenic aspiration-infiltrates, displaying—sometimes as early as 6 weeks after the onset of the infection—a remarkable size, dominate the clinical picture. On the other hand, in many instances—in older children and in young adults—in which large mediastinal lymph nodes suggested a late primary infection, and in which unmistakable pulmonary changes later developed, we actually deal with an initial lymph-node involvement. Nevertheless, it is not a lymph-node disease in the sense of Kourilsky and his associates, who assume the presence of a genuine exogenous primary, or post-primary, infection, in which the bacilli affected first the parapulmonary lymph nodes. On the contrary, in most of these cases *the reactivation of old tuberculous foci in lymph nodes occurred* and correspondingly, the enlargement of lymph nodes was the first clinical manifestation, while infiltrates of the pulmonary parenchyma appeared at a later stage of the cycle, following perforative bronchial lesions.

In a number of cases of true primary infections characterized by extensive caseous lymphonodo-bronchogenic aspiration-infiltrations, or, perhaps, by the presence of cavitary disintegration, we were unable to find a typical primary focus, even after careful search. We assume that in these observations the primary focus was surrounded and concealed by the necrotic aspiration-infiltration, or, that the primary focus was, together with the aspiration-infiltration, subjected to dissolution.* The correctness of this explanation is proved by cases in which a typical caseous primary focus remained visible, although enveloped in a necrotizing rubber eraser-like aspiration-infiltration, or was located approximate to a caseous aspiration-infiltration, but separated from it. (Figs. 4 and 5; see also Fig. 4 of our paper in Vol. 74).

In clinical descriptions we often encountered cases in which a rela-

* See Fig. 5 of our paper in Vol. 74 of the Arch. of Pediatrics.

Fig. 6. *Rubber eraser-like* ("regressive" "desquamative" "epituberculous") *pneumonia caused by hematogenic dissemination.*

The left lung of the case, demonstrated in Fig. 5. This lung displays only changes induced by hematogenic dissemination. Large, centrally necrotic miliary tubercles were observed microscopically. A reddish-gray, fleshy, rubber eraser-like homogenous infiltrate, filling the ("interfocal") spaces between miliary tubercles (intensely developed in the right lung also) predominated the changes of the left lung. The interfocal condensation microscopically was characterized by an intra-alveolar accumulation of large round cells ("alveolar histiocytes"). Disintegrating necroses, involving miliary tubercles and areas of the interfocal infiltration, produced very numerous parenchymal defects, (cav) which, when fully developed and cleansed, may mistakenly be considered as emphysematous bullae.

A pathogenic interpretation will be given in our next paper which deals with regressive ("epituberculous") pulmonary infiltrations.

Explanation of the signs see page 472.

tively large intrapulmonary calcium focus and calcified lymph nodes situated at the same level were considered as the remains of a primary complex which had appeared in childhood. It was often emphasized that the development and the continuous regression of these foci could be observed for decades. Indeed, we had the opportunity to investigate anatomically a series of cases of this kind, and to show that those intrapulmonary calcium foci constitute the sequelae of healed aspiration-infiltrations, and that the calcified lymph nodes are connected with typical perforative scars of the bronchi leading to the parenchymal calcium deposits. We wish to recall once more that scars of primary foci, as early as 4 years after the occurrence of an infection, often can only be detected by post-mortem radiography, and that we may assume that a primary focus can disappear tracelessly in a surprisingly short lapse of time.

We added in this paper to the traditional description of a classical primary complex in young children the regular occurrence of lymphonodogenous bronchial lesions and the observation of aspiration-infiltrations. Both phenomena are linked to the degree of the actually prevailing inflammatory disposition: *The higher the specific sensitivity is, the larger the penetrating and perforating lymph nodes, and the more voluminous the bronchial defects will appear.* On the other hand, the extension of the aspiration-infiltrations, too, is characteristic of the actually ruling inflammatory susceptibility. These influences are clearly recognizable in another important phenomenon in primary tuberculosis, caused by hematogenic dissemination of tubercle bacilli. *Hematogenic spread is an occurrence, which compulsively proceeds in every case of active tuberculosis.* It is present as early as 3 weeks after the onset of the infection. (Figs. 1-7) The higher the inflammatory disposition grows, the larger the miliary tubercles emerge, and the more the necroses prevail in them —just as in aspiration-infiltrations. Another important change dependent on increasing inflammatory disposition is the infiltration of the pulmonary tissue surrounding miliary tubercles ("interfocal infiltration"). At the climax of the inflammatory susceptibility vast, diffuse, seemingly homogenous pulmonary infiltrates of hematogenic origin may fill both lungs, (Figs. 6 and 7) reminiscent of alterations characteristic of animals which perish after intravenous injections of virulent tubercle bacilli.* The typical effects of an increased post-

* See Fig. 3 of our paper "On the Nature and the Pathogenesis of Some Pulmonary Changes which are Considered to be Particularly Characteristic of Tuberculosis in Childhood. I. Experimental Basis for a Concept of Pulmonary Tuberculosis in Man." Arch of Ped., Vol. 75, p. 315, 1958.

Fig. 7. *Pulmonary miliary tuberculosis with predominating caseous interfocal infiltration.*

The left lung of an eight-year-old girl, who died of tuberculous meningitis and miliary tuberculosis. (A. 980/31, Frankfurt-Main). The right lower lobe showed a typical, subpleural caseous primary focus, accompanied by considerably enlarged, caseated hilum lymph nodes. The parapulmonary lymph nodes of the left lung were not involved! Rather large miliary tubercles were dispersed in great numbers within the entire left lung. Their macroscopic visibility was, however, impaired by a very extensive, diffusely necrotic interfocal infiltration.

Explanation of the signs see page 472.

infectious resistance also manifest themselves very characteristically in both the aspiration-infiltrations and the miliary tuberculosis.

Comment

A tuberculous primary infection induces the development of a typical pathologic-anatomical entity. These changes, if they occur in a small child and the germs penetrate through the lungs into the organism, may appear suitable to prove that we really deal with the consequences of a first infection. The pulmonary primary complex always presents the same aspect in small children: It is characterized (1) by a relatively small pulmonary initial focus, located subpleurally, which crosses the acme of its development approximately 4-5 months after the onset of the infection and may heal in a further 4-5 years without leaving any trace. (2) A tubercular involvement of the regional hilum lymph nodes is present, at first characterized by their intense enlargement, later by necrosis (caseation) and disintegration. Systematic investigations showed that the lymph-node disease, starting from the first-involved hilum lymph nodes, rapidly spreads; particularly significant is the swift extension of the lymphnode tuberculosis to the right lung, in cases in which the primary complex is located on the left; while the lymph-node involvement, in general, remains restricted to the right side if the primary focus develops in the right lung. Six to eight weeks after the occurrence of the infection, the disintegrated lymph nodes of the primary complex begin to penetrate and to discharge into the tracheobronchial system—which is another important phenomenon of primary tuberculosis.

These observations permit the explanation of certain cases which repeatedly were interpreted as atypical primary infections. We too, formerly have considered pulmonary changes—which in reality are lymphonodo-bronchogenic condensations—to be "atypical" or "giant" primary foci, sometimes in cases in which the initial focus was enveloped by an aspiration-infiltration. "Primary focus cavities" develop, not through the softening of a primary focus, but by the transformation of an aspiration-infiltrate which before its disintegration may have incorporated the initial focus. Lymphonodogenous bronchial lesions may be mistakenly assumed to be "bronchial primary foci" in cases in which the initial pulmonary infiltrate is located in the proximity of the hilum, near to the discharged lymph nodes.

A new primary focus is not large enough to be—even in small

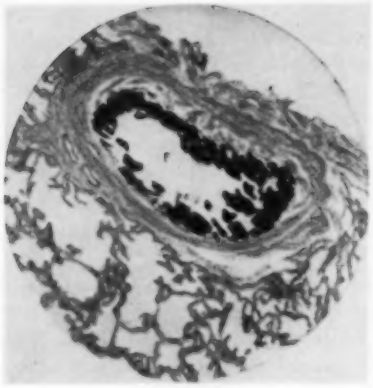


Fig. 8*a* and *b*. *A four-year-old boy died of pneumonia and pleuritis* (A. 578/30, Frankfurt-Main). The postmortem radiographie of the lungs disclosed a minute, hard, calcified focus (F), the scar of primary focus, located at the lingular tip.

a . . . . radiographic aspect of the minute primary focus.

b . . . . microscopic aspect of the primary focus.

No other tuberculous pulmonary changes were detected in this case. It is noteworthy that *no calcification in hilum lymph nodes was present*. (It should be emphasized that, today, we would investigate a similar case more thoroughly, looking for hidden tuberculous foci in abdominal and thoracic lymph nodes.)

Assuming that this child was infected shortly after birth, we understand that less than four years sufficed to make disappear, almost completely, the remnants of a primary focus! However, the child, perhaps, was contaminated later, which implies that the cicatrization process was even of shorter duration.

children—always detectable radiologically: Condensations larger than a cherry or even than a plum indicate lymphonodo-bronchogenic aspiration-infiltrations and not primary foci. The extension of infiltrates and cavities considered as atypical primary foci, results not from a particularly large quantity of inhaled germs, but is determined by the degree of the inflammatory disposition prevailing at the occurrence of the endogenous, lymphonodo-bronchogenic superinfection. Tubercular lymph nodes, originally intensely enlarged, may by discharge into the tracheobronchial system, disappear without leaving any trace.

A small, roundish, perhaps concentrically-structured, caseated, subpleural pulmonary infiltration occuring in small children may obviously indicate an initial focus of a primary infection. However, perfectly similar foci, accompanied by typically enlarged tuberculous lymph nodes also occur in older children and in adults of all ages, under conditions which permit definite exclusion of a primary infection. We may deal, in these cases, with the results of a new exogenous contamination which occurred after the biological healing of a previous tuberculosis ("post-primary start complex"). It is, however, much more common to encounter the results of the exacerbation of an old lymph-node tuberculosis in these cases, inducing bronchial lesions and consequently the appearance of the initial changes of a new tuberculous attack, i.e., the forming of an *early infiltrate resembling ("Postprimary start focus") start focus of childhood primary tuberculosis.*

Thus, we assume that the preliminary condition of the development of a pulmonary initial infiltration displaying the attributes of a typical primary focus of child pathology, is not its appearance in a pulmonary tissue previously spared by tubercle bacilli. We consider the following factors to be decisive: (1) the occurrence of a minimal infection and (2) the involvement of an organism, which shows no increase of the resistance bound to immune substances, and is characterized by a normal inflammatory disposition to tubercle bacilli. These conditions occur, not only in small children who yet had no opportunity to be infected, but also in persons hiding in their organism old, inactive tubercular foci. These observations agree with the results of our experimental investigations, showing that the deficiency of a particular resistance, or the absence of hypersensitivity to tubercle bacilli, may occur in organisms which were spared by a specific infection, but that both of these factors, so important in the development of pulmonary tuberculosis, also may become exhausted.

Fig. 9. *Healed primary complex in a 6½-year-old girl who died of diphtheria* (A. 961/30, Frankfurt-Main). A minute hilar calcium focus (arrows!) could be detected by postmortem radiography of the lungs, which, as histologic examination disclosed, was the encapsulated, calcium incrustated scar of an evidently entirely extinct tuberculous lymph node focus. *No trace of a pulmonary primary focus was present.* Hilum lymph nodes of both lungs contained caseated tubercular foci.

Summary

Every exogenous, new infection caused by inhalation, occurring in persons previously spared from tuberculosis, induces the formation of a very characteristic pulmonary focus, regularly accompanied by the tuberculous involvement of intensely enlarged lymph nodes. This pulmonary primary focus, at the culmination of its development, is, together with its perifocal infiltrate maximum cherry-sized. All condensations larger than the volume of a cherry —at any rate, larger than a plum—are not primary foci but lymphonodo-bronchogenic aspiration-infiltrations.

Within four years after the onset of a tuberculous contamination, the cicatrix of a primary focus grows so small that it can be detected only by post-mortem radiography. Foci resembling typical primary

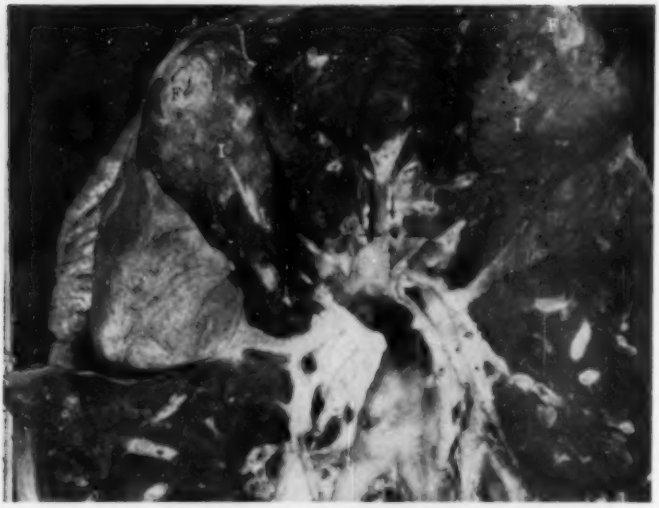
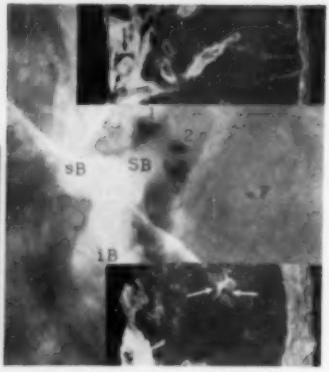


Fig. 10*a* above
Fig. 10*b* at right
Fig. 10*c* below



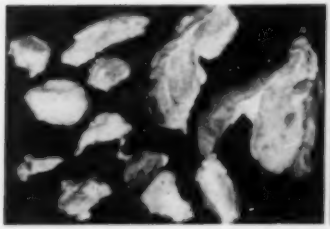


Fig. 10*a, b, c. Subapical early infiltrate* (F) *of the left lung resembling primary focus, accompanied by intense tuberculous involvement of the hilar lymph nodes.* ("Pseudo primary complex").

a . . . . roundish, markedly stratified caseous subpleural focus, (F) thoroughly associated with a "perifocal" aspiration-infiltration (ai). This perifocal condensation was wedge-shaped; the point of the wedge indicated a small perforative defect (arrows!) of the posterior oblique bronchus.

b . . . . Postmortem radiography of the left lung displaying a minute calcium focus (white arrows) of the apex of the lower lobe and two slightly larger hilar calcium foci (black arrows). Histologic examination disclosed cicatrices of entirely extinct tubercular foci.

c . . . . Enlarged hilum lymph nodes of the left lung affected by fresh caseous tuberculosis. The involved lymph nodes together formed an egg-sized lump.

infiltrates may occur in adults and in children, following perforative bronchial lesions induced by exacerbated tubercular processes in lymph nodes.

The presence of a so-called "primary focus" in the lungs does not mean that an organism contacted tubercle bacilli for the first time, but that at the time of the contamination no increase of the inflammatory disposition was present, and that the phlogogenic qualities of the germs were not diminished by reactive immune substances. The developments characterizing primary tuberculosis include the discharge of tubercular lymph nodes into the tracheobronchial system and the appearance of pulmonary aspiration-infiltrations.

The consideration of experimental observations promotes our knowledge related to pulmonary changes which occur following primary infection in children. Those two factors—the postinfectious increase of the inflammatory disposition and the appearance of an effective immunity—which in animal experiments decisively influenced the course of a tubercular disease, also are of profound formative influence in changes occurring in primary tuberculosis.

BIBLIOGRAPHY

Albrecht, H.: Über Tuberkulose des Kindersalters. Wien Klin. Wschr., 22:327, 1909.

Bieling, R. et Schwartz, Ph.: Über Immunitätsphänomene bei experimenteller Tuberkulose. Verh. Deutsch. Path. Ges., 25:334, 1930.

Blacklock, J.W.S.: Tuberculous Disease in Children: Its Pathology and Bacteriology. Med. Res. Coun. Sp. Rep. Ser., No. 172, London, 1932.

Blacklock, J.W.S.: The Epidemiology of Tuberculosis. Brit. Med. J., 1:707, 1947.

Canetti, G. et Robert, M.: La réinfection exogène chez le tuberculeux. Rev. de la Tuberculose, 14:451, 1950.

Canetti, G.: Exogenous reinfection and pulmonary tuberculosis. Tubercle, 31:224, 1950.

Fourestier, M., Bidermann, M. et Ettedgui, S.: A propos des rares manifestations ganglio-pulmonaires après vaccination par le BCG. La Presse Médicale, 59,443-444, 1951.

Ghon, A.: Der primäre Lungenherd bei der Tuberkulose des Kindes, Wien Berlin, 1912.

Ghon, A. et Kudlich, H.: Gibt es eine tuberkulöse primäre Infektion ohne Veränderung der regionären Lymphknoten? Arch. per le Scienze Med., 50:395, 1922.

Ghon, A.: Über Sitz, Grösse und Form des primären Lungenherdes bei der Säuglings- und Kindertuberkulose. Virch. Arch., 254:734, 1925.

Goldschmied, E.: Zur Frage des genetischen Zusammenhanges zwischen Bronchialdrüsen und Lungentuberkulose. Frankf. Zeitschr. Path., 1:332, 1907.

Hervouet, H.: Des adénopathies similaires chez l'enfant. Thèse de Paris, 1877.

Ickert, F.: Über exogene Reinfekte und die Superinfektion bei Tuberkulose, Leipzig, 1939.

Kourilsky, R. et Fourestier, M.: Deux accidents radiologiquement et cliniquement identiques à une primo-infection tuberculeuse, chez un enfant, à dix ans d'intervalle. Revue de la Tuberculose, 12:411, 1948.

Kourilsky, R., Bidermann, M. et Ettedgui, S.: Etude radio-tomographique et bronchoscopique de 71 cas de primo-infection tuberculeuse. Revue de la Tuberculose, 15:817, 1951.

Kourilsky, R., Bidermann, M. et Ettedgui, S.: Etude critique de la conception du complexe primaire dans la primo-infection tuberculeuse. Revue de la Tuberculose, 15:917, 1951.

Küss, G.: De l'hérédité parasitaire de la tuberculose humaine. Thèse de Paris, 1898.

Parrot, J.: Recherches sur les relations qui existent entre les lésions des poumons et celles des ganglions trachéo-bronchiques. C. R. et Mém. Soc. Biol., 308, 1876.

Ranke, K. E. v.: Primäre, sekundäre Tuberkulose des Menschen. Deutsch. Arch. f. klin. Med., 119:201, 1916.

Ranke, K. E. v.: Gesammelte Schriften, Berlin, 1928.

Redeker, F.: Über die exsudativen Lungeninfiltrierungen der primären und sekundären Tuberkulose. Beitr. Klin. Tbk., 59:588, 1924.

Schürmann, P.: Der Primärkomplex Ranke's unter den anatomischen Erscheinungsformen der Tuberkulose. Virch. Arch., 260:664, 1926.

Schürmann, P.: Zur Frage der Gesetzmässigkeiten im Ablauf der Tuberkulose unter besonderer Berücksichtigung der Entwicklungslehre Ranke's. Beitr. path. Anat., 81:568, 1929; 83:551, 1930.

Schwartz, Ph.: Empfindlichkeit und Schwindsucht, Leipzig, 1935.

Schwartz, Ph.: Die automatische, endogene, lymphadenobronchogene Reinfektion in der Initialperiode der Tuberkulose. Folia Pathologica, I, Springer, Wien, 1948.
Schwartz, Ph.: Neue Beiträge zur Morphologie und Pathogenese der Lungenschwindsucht. Folia Pathologica, II, Springer, Wien, 1952.
Schwartz, Ph.: Entzündung, Entzündungsbereitschaft und Immunität. Springer, Wien, 1953.
Schwartz, Ph.: Lymph node Tuberculosis: A Decisive Factor in Pulmonary Pathology. Archives of Pediatrics, 74:159, 201, 1957.
Schwartz, Ph. et Bieling, R.: Die Überempfindlichkeit bei einfach und doppelt mit Tuberkulose infizierten Tieren in ihrer anatomischen Auswirkung. Verh. Deutsch. Path. Ges., 26:226, 1931.
Schwartz, Ph., Rőszler, R. und Arkayin, C.: Über tuberkulöse, post-primäre Startkomplexe. Schweiz. med. Wschr., 72:141, 1942.
Simon, G.: Über die Früherscheinungen der kindlichen Lungentuberkulose. Beitr. zur Klinik der Tuberkulose, 59:529, 1924.
Simon, G.: Diagnostik und Klinik der Lungentuberkulose der Kindesalters, Stuttgart, 1940.
Simon, G. et Redeker, F.: Praktisches Lehrbuch der Kindertuberkulose, Leipzig, 1930.
Sloof, J.F.: Der tuberkulöse Primärkomplex im Röntgenbilde. Tuberk. Bibl., Leipzig, no. 63, 1937.
Straub, H.: Die tuberkulösen Rundherde der Lungen. Z. Klin. Med., 121:515, 1932.
Sweany, H.C.: Age Morphology of Primary Tubercles. Springfield, Ill., 1941.
Sweany, H.C.: Studies on the Pathogenesis of Primary Tuberculous Infection. I. The Regressive Lesions. Am. Rev. Tuberc., 27:559, 1933.
Sweany, H.C.: The Pathology of Primary Tuberculous Infection in the Adult. Am. Rev. Tuberc., 39:236, 1939.
Terplan, K.: Anatomical Studies on Human Tuberculosis. Am. Rev. Tuberc., 42, Suppl. No. 2, 1940.

EXPLANATION OF THE SIGNS IN THE FIGURES

We employ for the abbreviation of the names of the pulmonary lobes, the tracheobronchial system, the paratracheal and parabronchial lymph nodes, the vessels, and the parapulmonary spaces the following symbols:

A aorta; *P* pulmonary artery; *sc* subclavian artery; *S* upper lobe; *M* middle lobe; *Li* lingula; *I* lower lobe; *BD* right main bronchus; *BS* left main bronchus; *BT* stem bronchus; *b* bronchus in general.

Bronchi of the upper lobes.

sB main bronchus of the upper lobe; *hap* common stem of the axillo-pectoral system; *haa* ant. horizontal axillary bronchus; *hp* pectoral bronchus; *hap* post. horiz. axillary bronchus; *hd* horizontal dorso-paravertebral bronchus; *oa* ant. oblique bronchus; *op* post. oblique bronchus; *va* ant. vertical bronchus; *vp* post. vertical bronchus.

Bronchi of the (right) *middle lobe.*

mB main bronchus of the middle lobe; *ma* anterior branch; *mp* post. branch.

Bronchi of the lingula.

lB main bronchus of the lingula; *ma* ant. branch; *mp* post branch.

Bronchi of the lower lobes.

iB main bronchus of the lower lobe; *ds* superior dorsal bronchus of the lower lobe; *dd* descending branch of the sup. dorsal bronchus; *m* median bronchus; *a* axillary bronchus; *ac* paracardial branch of the axillary bronchus; *di* inferior dorsal bronchus; *bl* lateral basal bronchus; *bc* central basal bronchus; *bm* medial basal bronchus.

The parapulmonary spaces.

TBP intertracheo-broncho-pulmonary space; *B* bifurcation space; *Is* superior lateral interlobar space of the right lung; *Ii* inferior lateral interlobar space of the right lung; *Il* lateral interlobar space of the left lung; *Imd* medio-dorsal interlobar space..

Lymph nodes.

L, l lymph nodes in general; *TL* paratracheal lymph nodes; *Ly* paratracheal lymph nodes, located below the right subclavian artery; *sL* lymph nodes of the superior lateral interlobar space of the right lung; *sLs* supra-arterial lymph nodes of the sup. lat. interlobar space of the right lung; *sLi* infra-arterial lymph nodes of the sup. lat. interlobar space of the right lung; *iL* lymph nodes of the inferior lateral interlobar space of the right lung; *lL* lymph nodes of the lateral interlobar space of the left lung; *mL* lymph nodes of the medio-dorsal interlobar space; *mLs* supra-arterial lymph nodes of the medio-dorsal interlobar spaces; *mLi* infra-arterial lymph nodes of the medio-dorsal interlobar spaces; *BL* bifurcation lymph nodes; *BLd* right bifurcation lymph nodes; *BLs* left bifurcation lymph nodes.

Branches of the pulmonary artery.

The same symbols as for the concurrent bronchi, only in the place of *B* or *b*, *A* or a: (for example; main artery of the middle lobe: *mA*; or, axillary artery of the lower lobe: *aa*).

DEAFNESS PREVENTION IN CHILDREN

Loss of Hearing for High Tones

Edmund Prince Fowler, Sr., M.D.*
and
Eleanor C. Ronnei, M.A.**

New York

In the September issue of ARCHIVES OF PEDIATRICS, we published the first of a series of articles planned especially for the pediatrician, to better alert him to the necessity for the prevention, early detection and management of low tone deafness in babies and little children.

In the article which follows, we will discuss these items in relation to a specific pattern of neural deafness involving mainly the higher tones 2000 to 8000 cps—only. Unlike the well-known and more severe neural deafness involving all or most of the tones in the human hearing range, this kind of hearing loss is often completely missed or misdiagnosed in babies and young children at a time when its proper identification and management could be most helpful to the child's early speech-language and behavioural development.

In children, for several reasons, loss of hearing for the high tones only, is more rarely detected than low tone deafness. Foremost among these reasons is the fallacy that high tone deafness, commonly encountered in the aging, is rare in young children (therefore physicians rarely if ever test early for its presence). Second, there are often *no* physical symptoms to suggest that there is or has been a disease in the ear. Third, the child's response to many environmental sounds and to speech may seem to be similar to that of other children of comparable age. Finally, the pediatrician may believe that nothing can be done medically or surgically to improve the child's hearing. So he thinks he might as well ignore the condition.

All of the foregoing arguments are valid up to a point but stop short of the real truth. We hope to interest the pediatrician in extending his investigations beyond a seeming dead-end and into a better understanding of this type of hard of hearing child.

What symptoms should suggest a neural deafness rather than any one of the other types of deafness? Clues, we believe, are found

* Dr. Fowler is Chairman of the Subcommittee on the Hard of Hearing and the Deaf of the Council on Public Health and Education of the Medical Society of the State of New York.
** Mrs. Eleanor C. Ronnei is a consultant to the Subcommittee and Administrative Director of the New York League for the Hard of Hearing.

most frequently in slight deviations of a child's response to sound, in his speech-language development and social behaviour, in his health history, including prenatal and birth or delivery records, and, of course, in the results of special tests of hearing. Each of these clues may be difficult to track down because "children will be children" often unpredictable, inattentive but usually cooperative, eager and capable of giving you reliable information when efficiently managed. We suggest that you consider some of the following hearing response situations as the first line of attack.

A child with average normal hearing throughout the full frequency range will hear the sound of escaping steam, high-pitched whistles, or the squeak of a mouse, the whistle of a singing tea kettle, but the child with much tone deafness will not hear them. Both high tone deafened and normally hearing children will hear the sound of a foghorn, a boat whistle, an airplane overhead or a door slam equally loud. The child with the high tone hearing loss may hear the sound of a dog barking—but not his scratching at the door when he wants to come in. The child with normal hearing will hear men's and women's voices equally well, whereas the high tone hearing impaired child under consideration is likely to understand a man's voice relatively better than a woman's, because the former's is usually an octave lower in pitch. Furthermore, the young child with high frequency loss is apt to be frightened by loud sounds more frequently than the normally hearing child. This occurs largely because of the effect of the recruitment phenomena in neural deafness.

Both normally hearing children and those with high tone impairment may hear a call from another room, but the hearing impaired child is apt to ask, "What"? because he does not understand the articulation of the caller. Hearing impaired children are likely to shy away from situations in which they cannot compete successfully and are often called "poor sports" for that reason. We know a little kindergartner who refused to play in games when the starting signal was a high pitched whistle, but who participated happily when a low-pitched horn was substituted as the "GO" signal in the same game with the same children; an indication of high tone loss was revealed by his manner of response to the two differently pitched sounds. Such response pattern clues may in themselves seem inconclusive and should be substantiated by other attempts to isolate the reason for the child's play response.

High tone loss should also be suspected when a child's voice changes from good to a poor quality, when he speaks unnecessarily

loudly in an atmosphere of quiet or fails properly to regulate his voice to difference in background noise or when his voice lacks colorful inflection. Even little run-abouts like to hum or sing and manage a reasonable up and down melody pattern of sorts, an amusing and delightful sound that one seldom hears from children with severe high tone hearing loss.

Any reduction in either quantity or quality of speech output suggests the possibility of a hearing loss. A high tone loss is particularly suspected where the child seems to speak (articulate) reasonably well but fails to understand the speech of others. Pediatricians who recognize this fact will check both parrot-like repetition of words and their understanding in language form.

A careful study of the child's pre-natal, birth and health history may frequently give the pediatrician a clue to hearing loss by disclosing the presence of one of the many causes of high tone deafness. In many instances high tone deafness is congenital, having been acquired by the child when his mother had German measles or other virus infection in the first trimester of pregnancy. A prolonged delivery or a birth trauma resulting in severe anoxia can affect the child's membranous labryrinth and cause a loss of hearing, particularly in the high tones. Any drugs which produce anoxia may have little effect on the grosser tissues of the body but the ear, being the most sensitive organ, may suffer severely. Many children cannot tolerate quinine as is true of many adults. It causes a loud buzzing in the ears; this is not a real sound but an illusion of sound caused by irritation of the nerve apparatus of hearing. Since children usually fail to describe the conditions and doctors have no indication of need for withdrawal of the drug, permanent hearing damage can and often does result. There are several other drugs which act similarly if taken in sufficient amounts.

In the adult, mumps is frequently followed by a total deafness in one or both ears and there is no reason to believe that it does not affect children in the same way. When it affects only one ear it may not be detected because most people can function very satisfactorily with one ear, and this deafness may not be noticed until the child grows up and finds that he hears when he lies with one ear on the pillow but not with his other ear on the pillow.

It has been known for decades that meningitis may cause permanent loss of hearing; often a total loss in one or both ears. Yet in many cases deafness for the high tones only escapes suspicion even when the disease is recorded on the health history. High temperatures accompanying some undiagnosed disease or the various

exanthemata may be due to an undiagnosed meningitis occurring at the same time or following one of the childhood diseases.

Seldom found on the health record but of importance in tracking down the cause of a high tone hearing loss are accidents which cause acoustic trauma. A sudden loud noise close to the ear, especially if repeated, such as that produced by a vacuum cleaner, a power mower or a trumpet blast, may cause irreparable damage in the high frequencies. A fall or severe blow on the head can have the same kind of damaging effect. Sudden and severe deafness from such causes is usually accompanied or quickly followed by loud ringing in the ears, dizziness, nausea, and even vomiting. Again, because the child may be unable to put his sensations into words and because vomiting is common in children, the real problem escapes notice. Possible hearing involvement should be considered in all cases of accident to the head region. Even though the child does not lose consciousness, he may still have suffered a severe acoustic trauma—or even fracture of the skull.

While the pediatrician cannot prevent deafness from the causes described, or hope for cures when he has referred the case to specialists, he is responsible for the detection and reporting of these conditions. Furthermore, the pediatrician should bear in mind that all high tone deafness carries with it a potential for progressive loss. In Fowler's opinion, one of the most frequent causes of progressive nerve deafness is a lack of oxygen and other metabolites reaching the neural tissues in the inner ear and adjacent tissues. A lack of oxygen is often caused by a condition of the blood known as intravascular agglutination, or sludging of the blood. This phenomenon consists of a clumping of the blood cells into smaller or larger masses which stick together so tenaciously that they are pushed through the arteriolar capillary bottle-necks with difficulty so that the blood does not nourish the auditory tissues sufficiently to maintain them at their normal level of efficiency.

Sludging of the blood is caused by bacterial or virus infections, by shocks of various kinds and by long or repeatedly severe emotional episodes. The agglutinates are not clots in the proper sense of the word as they lack fibrin. Sludging is a common phenomenon and is a factor in several types of deafness, particularly sudden, severe or total neural deafness, the deafness accompanying old age and almost all forms of neural deafness. The otologist is beginning to better understand these disorders and to apply treatment and management conducive to prevention and reversal of the lesions.

What special tests of hearing are most useful in detecting high

tone loss in children? An audiometer is, of course, the best means at our disposal today. Standard techniques are usually successful for most otherwise normal children of four years of age or more. These tests are best administered by experienced and patient examiners and should be validated by several repetitions. For younger children and for those whose attention wanders too far and too often, a Peep Show technique has been devised. A useful test but far from perfect is the conditioned shock reaction or PGSR test (psycho-galvanic skin reaction method) which requires additional equipment and specially trained personnel. Available in some of the larger hospitals and speech and hearing centers, but seldom in the practising otologist's office, this test, supported by other hearing tests and trained observation of a perceptive clinician, has great value in the early detection of difficult to diagnose cases of high tone hearing loss.

Other useful diagnostic means such as the Galton whistle, used before Fowler and Fletcher introduced the pure tone audiometer, are still useful in determining the relative amount of hearing loss for high tones.

The pediatrician can also make use of a piano for this test. Although not usually found in a pediatrician's office, it can be used to determine whether or not the child hears both low and high pitched sounds equally well.

We often think of Ellen who taught her pediatrician this diagnostic trick. Ellen, two and a half years old, used the one word "BROKE" for everything that did not function as she thought it should. One day she carefully explained to her doctor that his piano was "broke" indicating the upper right hand area of the keyboard, but she said that it was O.K. over here, indicating the lower left and mid sections of the keyboard. Since then Ellen's doctor has suggested to his patient's parents that they use this musical instrument as a rough test of hearing acuity. In employing the piano, the parent should use the soft pedal and strike the keys at octave intervals. He should not let the child see him strike the keys and should observe the child's response to the different tones and intensities.

The pediatrician may also wish to suggest a screening speech hearing test for mothers to use in the home. A game of FIND-IT can be played, using objects or pictures known to the child. The mother can "hide" the object or its picture in plain sight. Then she can station herself at a given distance from the child and being sure that the child cannot read her lips, she can ask the child to find the spoon, the shoe, a block, a cookie, a doll carriage, or what not. Even

a very young child should be able to respond correctly at least fifty percent of the time. However, some children of better than average mentality, who have hearing limited to the low and low mid-tone, are able to put back the higher sounds mentally and to guess the spoken word almost as well as a normally hearing child. For this reason the pediatrician should suggest to the parent variations in the game and repetitions at different distances.

Many pediatricians may hesitate to refer children with mild to moderate neural deafness to otologists because they believe that nerve damage is irreversible and that their little patients will have to get used to this condition of "half hearing" by themselves. This is a totally unacceptable and defeatist attitude for a doctor to hold. Granted that there may be no medical or surgical help for the child, there are many sources of assistance for the child through education and knowledge of acoustics. Some can benefit from hearing aids; all can benefit from lip-reading, auditory and speech training. Many youngsters with high tone hearing impairment can learn to use their eyes to complement their hearing; they, for example, can see the lips speak the sound of F as in fire, and help the child to understand, though not hear, the difference between "Where is the fire?" and "Where is the tire?" Although his ear may never be able to monitor his articulation of "SH" as in Sheep and "CH" as in Cheap, he can learn to say them and use them correctly in communication with the help of a speech and hearing therapist.

Children with this type of hearing loss are educationally a most neglected group. They are not deaf enough "for all the world to see," nor do they hear well enough to get along without understanding guidance and educational help in the compensatory skills. Pediatricians might well expend effort in their behalf by encouraging Boards of Education to recognize the problem and to hire teachers for them. Certainly they will rarely require education in a special school or class, but they will need special help.*

The next article on deafness prevention in children will discuss deafness in the mid-tones or those most useful for speech.

* Good up-to-date literature for the pediatrician to use when talking to a Board of Education is found in HOW TO HELP THE HARD OF HEARING CHILD IN YOUR CLASSROOM, Ronnei, Eleanor C., (New York League for the Hard of Hearing, 480 Lexington Avenue, New York 17, New York), 15 pp., 25 cents.
THE CHILD WHO IS HARD OF HEARING, U. S. Children's Bureau, Folder No. 36 (Govt. Printing Office: Washington, D. C., 1952).
Where a hearing aid is desirable and the parent is unable to buy one, pediatricians should know of the aids provided by State Departments of Health.
For information on the non-medical resources available for hearing impaired patients, may we again suggest the American Hearing Society, 919 18th Street, N.W., Washington 6, D.C., and its chapters; The American Speech and Hearing Association, and your local and state Departments of Health and Education.
140 East 54th Street, New York 22, N. Y.
480 Lexington Avenue, New York 17, N. Y.

THE VENOUS CUTDOWN

EDWARD G. STANLEY-BROWN, M.D.*

New York

"The ratio of congenital absence of the saphenous vein is inversely proportional to the experience of the surgeon." This statement by Willis J. Potts (1) indicates one of the complications of the venous cutdown, that is tearing of the saphenous vein for this vessel is invariably present. Administration of parenteral fluids to pediatric surgical patients frequently requires a venous cutdown, a procedure which may present considerable technical difficulties and is often followed by both minor and serious complications. The technique to be presented is not original but has proven very satisfactory.

The number of accessible veins in a newborn is restricted and it is therefore vital to perform each cutdown correctly and care for it properly. If no ligatures are placed about the vein, the same vein may be used again after an interval of 10 days to 3 weeks when the incision in the vein wall has healed and the lumen re-canalized. This method obviates the slow healing and induration so commonly seen at the cutdown site from which it is frequently necessary to surgically remove the buried suture material.

The components of the cut-down set vary considerably but as a general rule include an imposing array of unnecessary equipment. The items we have found useful include:

1. Scalpel handle and #15 scalpel blade
2. 1 curved mosquitoe forcep (with dull tips)
3. 1 small straight forcep
4. 1 pair of toothed Halstead forceps
5. 1 pair of blunted curved manicure scissors
6. 1 skin hook
7. 1 pair of straight suture scissors
8. 4 strands of #00000 black silk (6 inches in length)
9. A plastic catheter with attached needle
10. 8-10 sterile gauze squares 2" x 2"
11. 1 small needle holder
12. 1 small curved cutting needle
13. Linen for draping or circumcision sheet
14. Balsa wood splint

* From the Surgical Service of St. Luke's Hospital in the City of New York.

15. 1″ and 2″ adhesive tape
16. 1% Procaine Hydrochloride Solution
17. #25 hypodermic needle
18. 1 2cc hypodermic syringe
19. Sterile gloves

The manicure scissors should be of the finest quality with the tips ground down sufficiently to remove the very sharp point.

The tip of the plastic catheter should be beveled and the very tip rounded off with scissors to avoid perforating the thin vein wall. The point should be ground off the needle at the hub of the plastic catheter.

All of these items can be autoclaved in one unit save for the scalpel blade, curved cutting needle, procaine solution and the surgeons gloves which should be separately prepared.

The fluid or blood to be administered should be at hand with the tubing attached and enough solution permitted to run through to remove air bubbles.

Effective restraint of the extremity is important. An ordinary bass wood splint is cut with heavy bandage scissors to extend from the upper one-third of the thigh to a point 2 inches beyond the sole of the foot.

The width of the splint will vary with the circumference of the patients leg and in general a new splint should be fashioned for each individual. The splint is well-padded with gauze fluffs which are secured with gauze roller bandage and adhesive tape. The patient's leg is then placed on the splint with the foot in eversion and the thigh encircled with two strips of 2″ adhesive tape. No tape is placed over the knee joint as this may occlude the saphenous vein. With the foot everted and at an angle of 90 degrees dorsiflexion two strips of one inch tape are placed crossing just below the medial malleolus thus firmly securing the foot. The now well restrained extremity is attached securely to the bed or operating table with adhesive tape.

A good source of light is adjusted and if the patient is restless a sugar pacifier provides the necessary calm.

The cutdown should always be undertaken with anesthesia. If the patient is about to undergo general anesthesia we prefer to do the cutdown under local and thus shorten the duration of inhalation anesthesia.

Following these preparations the skin of the ankle is prepared with antiseptic solution. The next step is to inject the site of incision with 1% Procaine Hydrocloride solution. Inducing local anesthesia

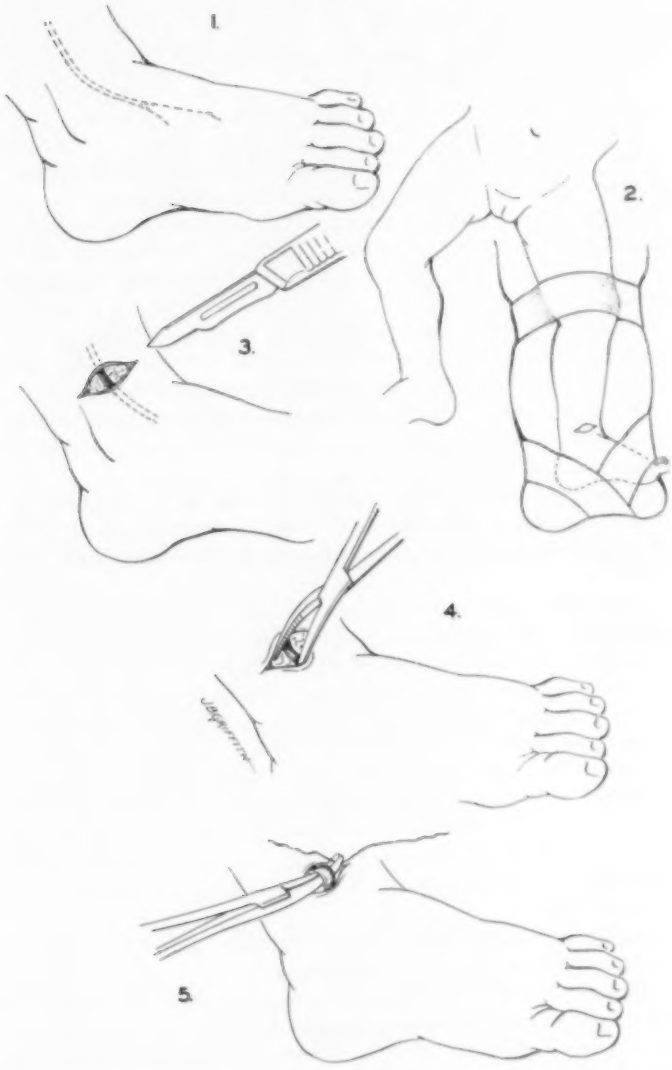


Fig. 1. The internal saphenous vein anterior to the medial malleolous.
Fig. 2. Method of immobilizing the foot on a padded splint.
Fig. 3. 1 centimeter long transverse incision through the skin only.
Fig. 4. Careful blunt dissection to isolate and elevate the vein.
Fig. 5. A single silk strand for traction purposes only.

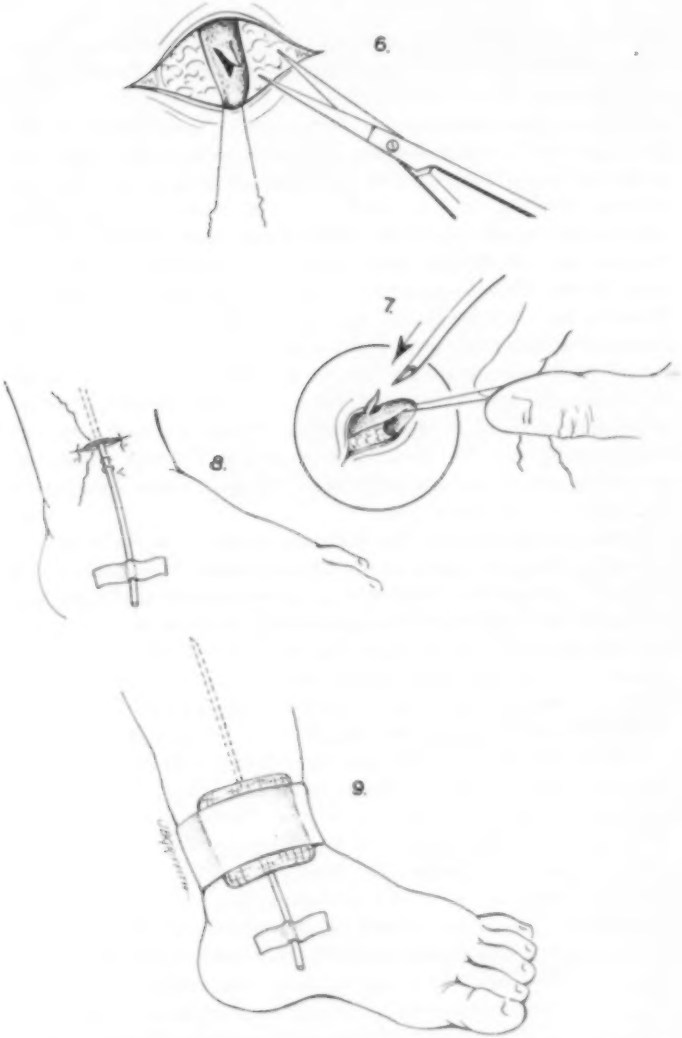


Fig. 6. With the vein held taut a "V" shaped nick is made with scissors.
Fig. 7. Maintaining tension the plastic catheter is threaded into the vein with the bevel down. The traction strand is withdrawn.
Fig. 8. The skin edges are approximated with interrupted silk sutures and a single suture is placed to affix the catheter to the skin surface.
Fig. 9. Having threaded the plastic catheter well up into the vein a firm pressure dressing is applied to the incision. No ligatures have been placed about the vein itself.

prior to donning gloves and draping the patient allows for an interval in which the anesthetic agent may take full effect. The operative area is appropriately draped with sterile linen.

The transverse skin incision slightly above and anterior to the medial malleolus should never exceed one centimeter in length and is carried through all layers of the skin. Bleeding is controlled by pressure with a gauze pad and the edges of the incision are spread apart with a curved clamp. By means of this blunt spreading dissection the vein will appear, bluish in color, at the depths of the incision. A skin hook serves well as a retractor for the skin and is preferred to grasping the lips of the wound with forceps. Still using the curved forcep the areolar tissue on either side of the vein is spread apart along the long axis of the vein. Once this bilateral mobilization of the vein is complete the forcep may be readily passed underneath the vein. This step will be difficult and may result in tearing of the vein if it has not been adequately freed up. Using the curved clamp a strand of #00000 black silk is drawn under the vein and serves as a means of traction.

The actual incision into the vein wall must expose the lumen by not more than one third of its circumference. Too shallow an incision will result in threading the catheter into the adventitia and too deep a cut will invariably cause tearing or actual division of the vein during canulation. Holding the vein taut with the silk traction strand a "V" shaped nick on the anterior wall is made with the manicure scissors, the curved blunted blades facing downwards. A backflow of blood indicates that the vein lumen is open.

Still keeping the vein under tension with the encircling silk strand the plastic catheter is held with the bevel facing down and the tip is gently insinuated into the lumen. Retrograde flow of blood into the catheter assures the operator of its proper position. The infusion tubing is promptly attached and fluid permitted to run in at once since any delay may allow clot formation.

Once the infusion has started the catheter is positioned with the tip three to four inches proximal to the skin incision and the traction strand is withdrawn. It is well to suture the catheter to the skin distal to the incision in order to prevent inadvertant removal during closure of the skin incision which is accomplished with two or three interrupted sutures of #00000 black silk approximating the skin edges.

The extremity may now be removed from the balsa wood splint and a firm pressure dressing applied over the incision. As a further precaution the plastic catheter is secured to the leg with adhesive

tape and the words "CUT DOWN" printed on the outer adhesive wrapping with a red china marking pencil. This simple expedient precludes mistaking the cutdown for a simple intravenous needle.

It is unnecesary to restrain these patients once the cutdown is completed, for if properly performed inadvertant removal is very difficult and the pliable catheter should not occlude on bending, as at the knee joint.

The plastic catheter may be safely left in place for three to four days and by adjusting the rate of flow the solution may be kept running continuously. When no longer needed the suture affixing the catheter to the skin is cut and the catheter simply withdrawn without disturbing the outer dressing. The skin sutures should be removed after six to seven days, a step often forgotten and brought to the surgeon's attention only at a subsequent office visit. If left in situ too long the catheter may result in thrombosis or thrombophlebitis. Infection at the cutdown site or along the course of the vein reflects some break in sterile technique during the performance of surgery.

Summary: We have presented a technique for venous cutdown, not original but satisfactory. The necessary instruments are listed and the procedure described in detail and illustrated. Ligatures about the vein itself are unnecessary and should be avoided.

REFERENCE

(1) Potts, Willis J.: Pediatric Surgery, J.A.M.A., 157:627, 1955.

862 Park Avenue

PORENCEPHALY AND COMPLICATED CEREBRAL PALSY*

SAMUEL ROSNER, M.D., F.I.C.S.

New York

Porencephaly, as defined by Heschl,[1, 2] is a defect in the cerebral substance in one or more places. The cerebral substance is lacking through the entire thickness of the brain. "There is a canal through the brain which begins on the outer surfaces of the brain and ends in the cerebral ventricles." Many possible causes have been proposed, destruction of brain tissue by occlusion of cerebral vessels, haemorrhage prior or associated with birth,[1, 2] encephalitis,[3] sometimes intrauterine.[4] Globus,[5] reported bilateral porencephaly in one case which showed signs of meningoencephalitis and occlusion of the pial vessels. Granulomatous infections such as syphilis[6] and tuberculosis[7] have also been described as possible causes. Jaffe,[8] suggested intrauterine or birth trauma as a cause. The trauma may be directly to the brain or circulatory affecting the large veins and their drainage. Marburg[9, 10] demonstrated porencephaly due to interference with venous drainage at birth or fetal life. LeCount[6] and Semerak, proposed that cystic brain defects were due to developemental defects in the germ plasm caused by abnormality or injury of the germ plasm before fertilization. They also thought that congenital vascular defects may also be a cause of cerebral cysts.

The preponderance of evidence points to the vascular pathology, either developmental, traumatic, infective or obstructive as the cause or combination of causes of porencephalic cyst formation. Venous pathology in many cases seems to have been the cause of this type of cyst.

The following case of complicated cerebral palsy,[11, 12, 13] due to bilateral porencephalic cysts is pertinent to the study of both cerebral palsy and porencephaly. Complicated cerebral palsy is defined as cerebral palsy with epilepsy, or mental defect or both.

Case History:—S.J.W., Female, child, age 17 months.

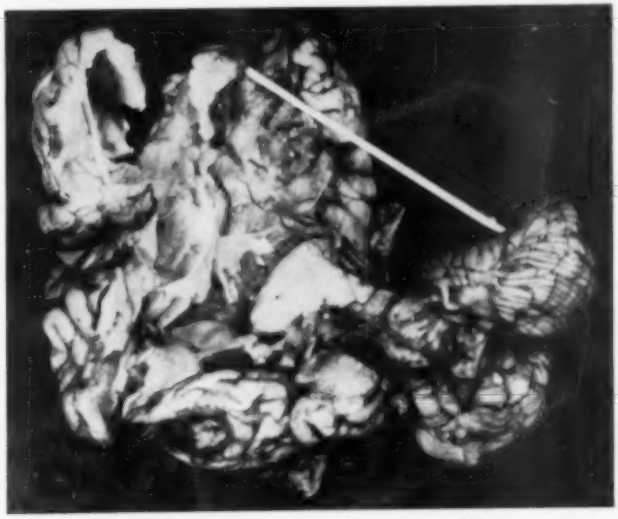
Past Illness:—Pneumonia in December, 1957.

History of Present Complaint:—The child was born November 1956, prematurely at 8 months. A report from Children's Hospital of Columbus, Ohio, stated that the child had been admitted March 18, 1957 with temperature fluctuating between 100 degrees to 104

* Samuel Rosner, M.D., F.I.C.S., Director of Neurology and Neurological Surgery, Parkchester General Hospital.

degrees. On physical examination there was a "hemangioma, or what appeared to be a hemangioma on the flat bridge of the nose." "The liver was two fingers below the costal margin. The head was 14 inches in diameter. She was unable to hold her head up, to follow light, or to smile. A diagnosis of brain damage, hypothalamic region; this might prove to be a case of cerebral palsy, hemangioma with questionable absent nasal bone." "Lumbar puncture was negative." E.E.G. was "grossly abnormal, strongly suggestive of severe organic disease of the brain with irritative lesion or scar in the right anterior area."

"Ventriculogram showed extreme dilatation of the right half of the ventricular system." X-ray of chest showed bronchiolitis.



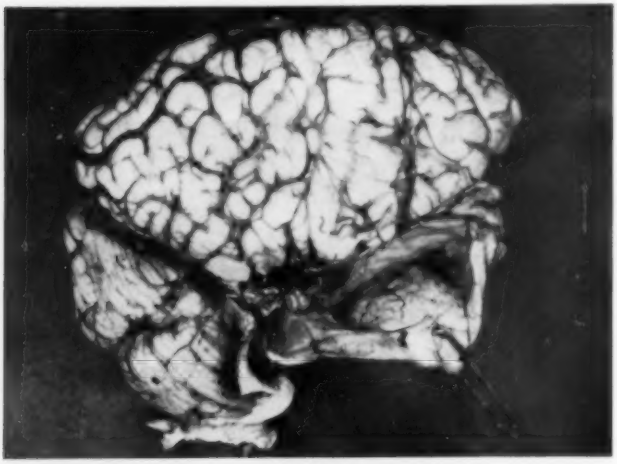
Massive Porencephalic Cysts Bilaterally

"*Final Diagnosis*:—Cerebral defect. Probably anterior meningocele. Mental retardation."

The child was seen by me on March 3, 1958. There was a history of convulsions since March, 1957. The convulsions became more frequent and more severe with the passage of time. The child was spastic in all limbs with inability to raise head. The hemangioma caused the right eye to be depressed. The diagnosis was mental defect (gross) cerebral palsy more marked on the left, epilepsy (severe) uncontrolled by medication and hemangioma of the nose connected through the skull with the brain.

March 5, 1958: —Right craniotomy revealed an enormous porencephalic cyst completely filling the right cerebral hemisphere. The cortex was thinned markedly and collapsed when the cyst was evacuated. The gyri were smaller than normal and gave a fluid thrill on palpation before evacuation of the cyst. The cyst contained about 100 c.c.'s of clear fluid. The temporal lobe seemed normal.

The child died of pneumonia seven days after operation.



Porencephalic Cyst In Temporal Lobe

Post-Mortem Examination of the brain:—Laboratory Report. Gross: The skull and meninges appear normal. The fontanelles are patent and are not bulging. The brain has been previously fixed and somewhat dissected. It measures 14 cms. in the occipital-frontal plane, 9 cms. from side to side and 7 cms. in height. The cerebellum, pons, and mid-brain show no gross lesions. The circle of Willis is intact. The vessels are thin and delicate throughout and the lumina are widely patent. The meninges show moderate congestion with no areas of hemorrhage or exudate. There is a moderate collection of subarachnoid fluid in some areas. There is no herniation of the uncinate gyrus at the base of the brain. Externally the gyri and sulci of the left cerebral hemisphere appear normal. The right cerebral hemisphere appears smaller in overall dimensions and the gyri appear considerably smaller on the left. Both hemispheres have a cystic consistency more marked on the right than on the left. The cut section of the left cerebral hemisphere shows the brain paren-

chyma to appear normal with the usual landmarks evident and the normal differentiation of grey and white matter. The ventricular system on the left appears somewhat greater than usual and there is some blunting of the anterior horn of the ventricular system. The right cerebral hemisphere shows marked diminution in the thickness of the brain parenchyma which varies from 4 to 8 mms. in thickness. In many areas the grey and white matter are not well demarcated. In some areas they are well demarcated and are markedly diminished in width. The residual brain substance on the right is firmer than on the left. There is a corresponding enlargement of the right ventricular system and there is slight thickening and congestion of the lining of the ventricular system on the right with some recent blood staining posteriorly. The choroid on the left shows cystic changes producing a grape-like configuration. The vein of Trolard is somewhat dilated on the right.

DIAGNOSIS:—Final Anatomical Diagnosis:

(1) Post-operative craniotomy, recent.

(2) Brain atrophy, marked, congenital, with hydrocephalus and cerebral palsy (clinical).

SUMMARY

The patient was a seventeen month old child suffering from cerebral palsy, gross mental defect and severe grand mal epilepsy.

The clinical history, operative findings and post-mortem description of the brain are presented.

** Carried out with the aid of Parkchester General Hospital Fund.

1. Heschl, R.: Gehirndefekt und Hydrocephalus, Vrtljschr. prakt. Heilk. 61:59, 1859.
2. Heschl, R.: Neue Falle von Proencephalie, Vrtljschr. prakt. Heilk. 100:40, 1868.
3. Strumpell, A.: Uber die acute Encephalitis der Kinder (Poliencephalitis acuta, cerebrale Kinderlahmung), Jahrb. Kinderh. 22:173, 1884.
4. Schultze, R.: Beitrag zur Lehre von den angebornen Hirndefecten (Porencephalie). Nach einer auf der 58. Versammlung deutcher Naturforfscher und Aerzte in Strassburg 1885 gemachten vorlaufigen Mittheilung, Heidelberg, C. Winter, 1886.
5. Globus, J. H.: Contribution to the Histopathology of Porencephalus, Arch. Neurol. & Psychiat. 6:652, 1921.
6. LeCount, E. R. and Semerak, C. B.: Porencephaly, Arch. Neurol. & Psychiat. 14:365, 1925.
7. Winterode, R. P., and Lewis, N. S. C.: Case of Porencephalic Defect Associated with Tuberculous Encephalitis, Arch. Neurol. & Psychiat. 10:302, 1923.
8. Jaffe, R. H.: Traumatic Porencephaly, Arch. Path. 8:787, 1929.
9. Marburg, O., and Casamajor, L.: Phlebostasis and Phlebothrombosis in the Newborn and in Early Childhood, Arch. Neurol. & Psychiat. 52:170, 1944.
10. Marburg, O.; Rezek, P. R., and Marks, M. B.: Porencephaly: II. Studies in Phlebothromobsis and Phlebostasis, J. Neuropath. & Exper. Neurol. 4:43, 1945.
11. Rosner, S.: The Pathology & Surgical Treatment of Complicated Cerebral Palsy. Arch. of Ped.—Vol. 75, No. 1, Jan. 1958, Pages 1-10.
12. Rosner, S.: Observations on the Pathology & Surgical Treatment of Complicated Cerebral Palsy. Parkchester General Hospital J. of Med. & Surgery, June, 1958, Vol. 4, No. 1, Page 17-24.
13. Rosner, S.: The Surgical Pathology of Complicated Cerebral Palsy. To be published in the near future.

Current Literature

By

MICHAEL A. BRESCIA, M.D.

KAUFMAN, H. AND ISORNI, P.: VARIATIONS OF THE BLOOD CHOLESTEROL LEVEL IN THE COURSE OF ATTACKS OF BOUILLAUD'S DISEASE. (Presse Médicale 65:600, March 30, 1957).

Five to ten days following the onset of clinical improvement from an attack of acute articular rheumatism, there occurs a marked rise in blood cholesterol. The total cholesterols could rise two to three times the normal. This hypercholesterolemia may last as long as several weeks. The authors studied the correlation between the cholesterol increases and the white blood count and sedimentation rat. The cholesterolemia level varies in inverse ratio to the sedimentation rate except for the end of the cholesterolemic phase during which the fall of the cholesterol level is consistent with the stability or increase of the sedimentation rate. Whatever may be the theoretical explanation of the phenomenon, its observation is very helpful in the appraisment of the potential evolution of acute articular rheumatism. M. A. B.

ORTHOPEDICS FOR THE GENERAL PRACTITIONER. By WILLIAM E. KENNEY, M.D. AND CARROLL B. LARSON, M.D., F.A.C.S. Cloth. Pa. 400. Illustrated. Price $11.50. St. Louis, Mo.: The C. V. Mosby Co., 1957.

The material in this book is drawn from the authors' extensive experience and from other reference sources. The well organized chapters and good summary tables of orthopedic problems render such information readily accessible for quick reference. The physician, in arriving at a diagnosis, has essentially two very powerful tools at his disposal: the complaint of the patient and the anatomic location about which the complaint is made. This book is so organized that the doctor can refer to that chapter dealing with the anatomic location pointed out by the patient (for example, the hip) and there find a list of usual complaints pertaining to such a location, with the likely diagnoses noted opposite the complaint; he will also find the page references to the description of these conditions, the ways and means of establishing a diagnosis and treatment. By using this book, a doctor should be able to arrive at a diagnosis and outline his treatment with a minimum loss of time. The book is intended for the family doctor or general practitioner and the

pediatrician. Orthopedic disturbances in children have certain aspects which are quite different from the principles obtaining in the adult. Orthopedic textbooks do not readily yield information with practical answers although they describe a condition quite adequately. To use a text means reading an entire book—something a physician hardly can do when faced with an immediate problem. "Orthopedics for the General Practitioner" does all this adequately, combined with minimal reading. There are 180 illustrations and photographs which are very helpful, and the clarity and presentation in reading is simple and precise. I heartily recommend this book to all doctors practicing orthopedics.

JOSEPH M. COVELLI, M.D.

WYNN, R. S.: RETINOBLASTOMA. REPORT OF A CASE AND REVIEW OF THE LITERATURE. (Journal National Medical Association 50:254 July 1958).

Next to malignant melanoma of the uveal tract, retinoblastoma is the most common intraocular tumor. It usually appears in children under 5 years of age, less frequent after 5 years and extremely rare after the 10th year. There seems to be no significance in sex or race distribution, however familial occurrence is a feature of retinoblastoma.

Healthy parents who have had one child with retinoblatoma may be advised to have more children but survivors of retinoblastoma should be advised not to have children.

Retinoblastoma may be confused with retrolental fibroplasia, persistant, hyperplastic primary vitreous, metastatic retinitis, Coat's disease, massive retinal fibrosis in children, endophthalmitis and medullated nerve fibers.

X-ray therapy is successful in many cases of retinoblatoma in which less than one third of the retina is involved. There is a higher incidence of serious complications with x-ray therapy than with radon seed or radium disks. Triethylenemelamine an analogue of nitrogen mustard has been used with irradiation to some success in the treatment. T.E.M. has an advantage in that it is given orally which facilitates its use in children.

A case report of a 13 month old child with retinoblastoma of the left eye who later had a similar tumor of the right eye which caused death has been given. I believe that in any advanced case of retinoblastoma the eye should be enucleated to prevent a possible metastisis and death even if it is the only remaining eye.

Author's Summary.

MARONDE, R. F.: CAUSE OF DEATH IN PERSONS WITH CONGENITAL HEART DISEASE. (American Journal Medical Sciences 236:41 July 1958).

The causes of death of 84 patients with congenital cardiac defects who had survived until 2 years of age or longer are reported. Patients with isolated interatrial septal defects more often than not die of an unrelated cause, even when the defect is large. Future evaluation of any surgical correction of this defect, unless cardiac symptoms are present, would be difficult. The patients with an isolated interventricular septal defect but without dextroposition of the aorta, had a cause of death unrelated to the defect, except for brain abscesses.

Author's Summary.

SPITZNAGEL, J. K.: EFFECTS OF CORTISONE AND ACTH IN MUMPS MENINGO-ENCEPHALITIS. (Annals Internal Medicine 49:61 July 1958).

The efficacy and safety of cortisone in the symptomatic treatment of four patients with severe or moderately severe mumps meningoencephalitis have been investigated. A fifth patient was treated with ACTH (corticotropin). Diagnoses were confirmed by lumbar puncture in all cases, and complement fixation titers were determined in four.

Cortisone was administered in doses of 200 to 300 mg./day (ages of patients from 20 to 37 years), and ACTH in doses of 60 mg./day. In all cases but one the drugs were discontinued by progressive decrements.

Cortisone and ACTH appeared to produce rapid remission of headache, nausea, anorexia, vomiting, photophobia, lethargy, delirium and nuchal rigidity. Fever, although it fell rapidly, tended to return to previous levels for short periods of time, accompanied by minor recurrences of other symptoms. Orchitis developed in one patient during cortisone therapy. This patient, and one other, suffered additional salivary gland involvement during therapy. Abrupt termination of therapy in one case after three doses of cortisone resulted in a dramatic recurrence of previous symptoms.

The duration of mumps in these patients was not shown to differ from that in patients not receiving cortisone or ACTH. No evidence of residual disease of the nervous system could be detected in any of the patients.

Author's Summary.

WHY RISK DELAYED RECOVERY FROM PYODERMAS?

Many of the organisms causing pyoderma are refractory to routine antibiotic therapy. If the offending organisms are resistant staphylococci, CATHOMYCIN (novobiocin) is indicated. CATHOMYCIN has an established record* of effectiveness against strains of organisms resistant to other antibiotics. It may be administered alone, or combined with other antibiotics for protection against the emergence of resistant strains.

Of particular value in hard-to-control pyodermas caused by resistant staphylococci, CATHOMYCIN is rapidly absorbed—producing therapeutic blood levels that last for 12 hours or more. The drug is generally well tolerated and there is no evidence of cross-resistance with other antibiotics.

CATHOMYCIN®

NOVOBIOCIN

for staphylococcic septicemia, enteritis, postoperative wound infections and other serious staph infections.

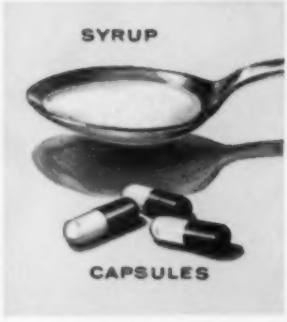
DOSAGE: Adults: CATHOMYCIN Sodium 2 capsules b.i.d. or CATHOMYCIN Calcium Syrup 4 teaspoonfuls b.i.d. Children: (up to 12 years) 2 to 8 teaspoonfuls daily in divided doses based on 10 mg. CATHOMYCIN per lb. of body weight per day.

SUPPLIED: Capsules sodium novobiocin, each containing the equivalent of 250 mg. of novobiocin—vials of 16 and 100—and as an orange-flavored syrup (aqueous suspension), in bottles of 60 cc. and 473 cc. (1 pint). Each 5 cc. CATHOMYCIN Syrup contains 125 mg. (2.5%) novobiocin, as calcium novobiocin.

*Complete bibliography available on request.

NEW

For Parenteral Therapy LYOVAC® CATHOMYCIN



MERCK SHARP & DOHME Division of MERCK & CO., Inc., Philadelphia 1, Pa.

The great operatic works of Rossini have been enjoyed by millions for many decades

THINGS THAT ENDURE

Good things endure . . . a work of art, a literary classic, a proud bridge . . . a dependable pharmaceutical. Such is **Desitin Ointment.** For over 35 years Desitin Ointment has endured as an incomparable, safe way to prevent and clear up diaper rash . . . and as a soothing, healing application in wounds, burns, external ulcers and other skin injuries.

Desitin®

DESITIN CHEMICAL COMPANY PROVIDENCE 4, R. I.